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LAYERS AND STRUCTURE OF EYEBALL

Basic Anatomy of Eyeball

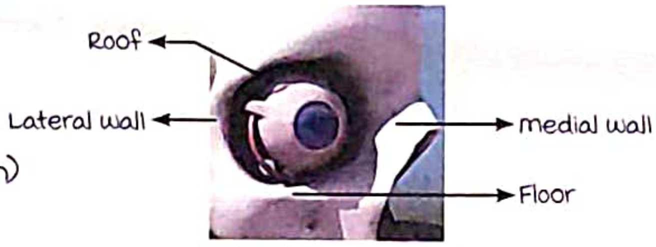
00:01:05

Shape : Aspherical (Ablate spheroid)

Volume : 6 ml

Site : Orbit (bony cage)

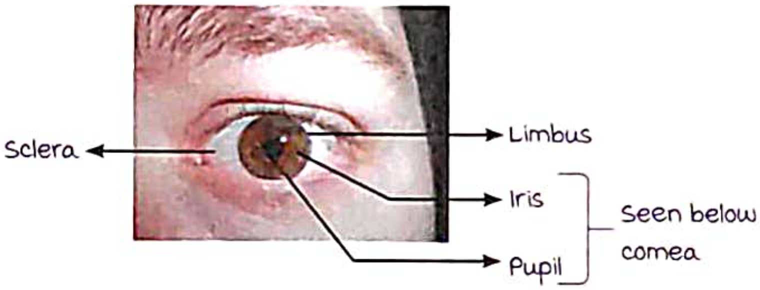
Axial length/Anteroposterior diameter : 24 mm (2.4cm)

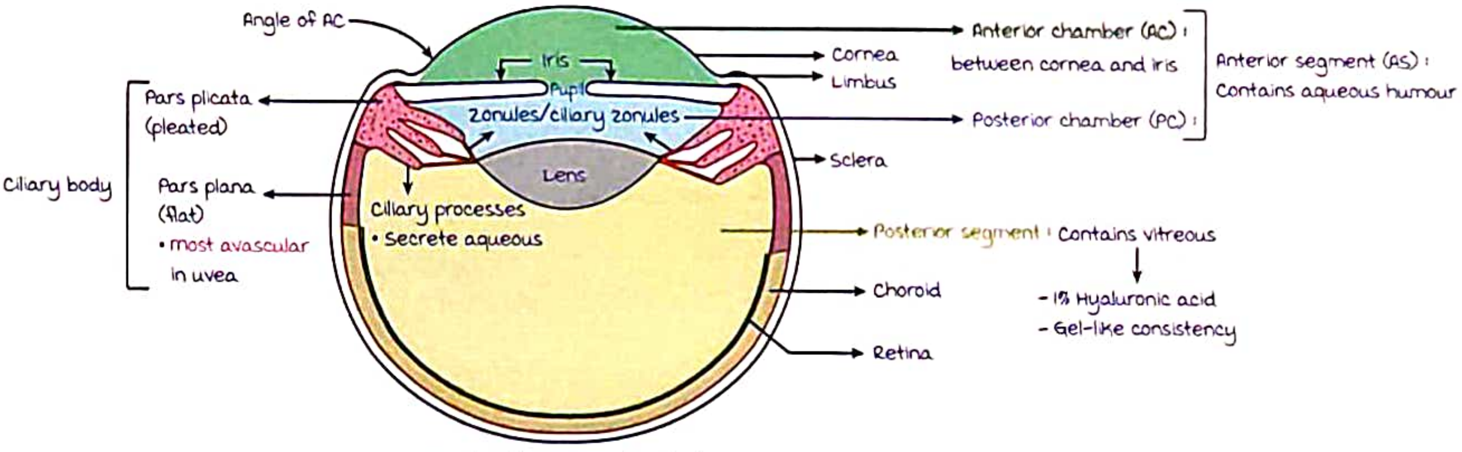

Roof
Lateral wall
medial wall
Floor

Orbit containing eyeball

Layers

- Outermost
  - Anterior 1/6th : Cornea
    - Transparent
    - Convex shaped
    - Refractive power present
  - Limbus
    - Corneo-scleral junction
  - Posterior 5/6th : Sclera
    - White (opaque)
    - Covered by conjunctiva (transparent mucous membrane)
- Middle : Uveal tract
  - Parts (Anterior & Posterior)
    - a. Iris
    - b. Ciliary body
    - c. Choroid
  - Vascular Layer
- Innermost : Retina
  - Neurosensory layer
  - Present only posteriorly


Sclera
Limbus
Iris
Pupil
Seen below cornea


Angle of AC
Iris
Pupil
Zonules/ciliary zonules
Lens
Ciliary processes
• Secrete aqueous
Ciliary body
Pars plicata (pleated)
Pars plana (flat)
• most avascular in uvea
Anterior chamber (AC) : between cornea and iris
Cornea
Limbus
Posterior chamber (PC) :
Anterior segment (AS) : Contains aqueous humour
Sclera
Posterior segment : Contains vitreous
- 1% Hyaluronic acid
- Gel-like consistency
Choroid
Retina

Structural anatomy of eyeball

----- Active space -----

Aqueous humour :

- Fluid secreted from ciliary processes
- Rate of secretion : 2.3 μL/minute
- Function : exerts Intraocular pressure (Iop)
  - measured by Tonometry.
- Aqueous outflow : 0.25 μL/min/mm Hg
  - Via angle of AC → Depth : 3 mm (2.5 mm – 4.4 mm)
  - measured by tonography

ANATOMY OF CORNEA, ANATOMY OF SCLERA AND ITS PATHOLOGIES

Neurovascular Supply of Cornea

00:00:30

Blood supply : Avascular ⟶ Cornea : Transparent

Relative index : 1.376

Refractive power : +43D to +48D

(Converging power)

Nerve supply (Sensory) :

Trigeminal → Ophthalmic → Nasociliary → Cornea

Corneal Blink Reflex : Test of corneal sensation.

Stimulus : Touch cornea with cotton wisp.

↓

Afferent (sensory) : Trigeminal nerve (CN V)

↓

Trigeminal nucleus

↓

Facial nucleus

↓

Efferent : Facial nerve (CN.VII) —✗→ Bell's palsy

(motor)

Inability to close eye : Lagophthalmos

Effector : Orbicularis oculi

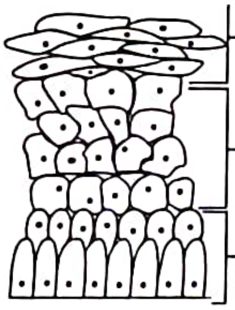
↓ No blinking ⟶ Corneal anaesthesia

Response : Blinking.

Causes :
- Herpes keratitis (m/c)
- Diabetes
- Leprosy
- Topical medications (ex. Timolol)
- Acanthamoeba keratitis
- Contact lens wear
- Surgical trauma

Layers of the Cornea

00:15:00

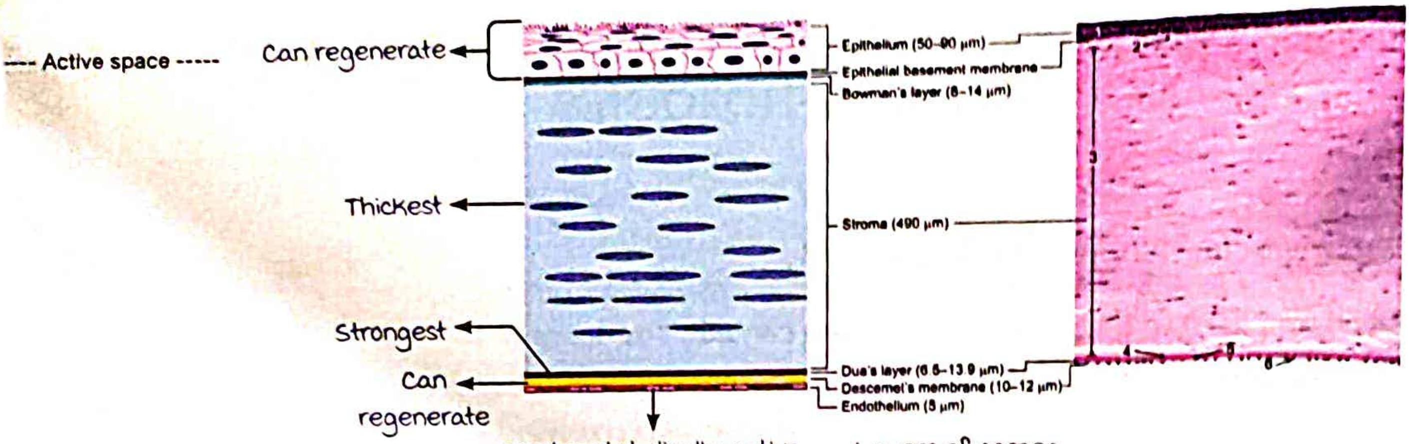


→ Superficial layer
- Squamous cells have microvilli → Adhesion of tear film

→ middle layer
- Wing-shaped cells

→ Basal Layer
- Columnar cells → can undergo mitosis / regenerate

Corneal epithelial histology


---- Active space -----
Can regenerate
Thickest
Strongest
Can regenerate
most metabolically active
Epithelium (50–90 µm)
Epithelial basement membrane
Bowman's layer (8–14 µm)
Stroma (490 µm)
Dua's layer (6.5–13.9 µm)
Descemet's membrane (10–12 µm)
Endothelium (5 µm)
1
2
3
4

Layers of cornea

1. Anterior (Outermost) Layer – Epithelium :

Histology : Non-keratinized stratified squamous epithelium.

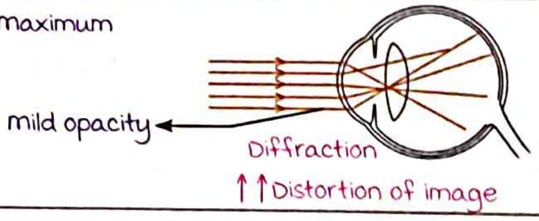
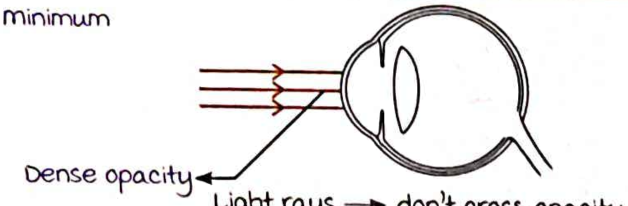
Note : with respect to the centre of the eyeball.

	Outermost layer	Innermost layer
Cornea	Lies anteriorly	Lies posteriorly
Retina	Lies posteriorly	Lies anteriorly

2. Bowman's membrane :

- Not a true basement membrane → not stained by PAS (Periodic Acid Schiff reagent)
- Acellular → Cannot regenerate → Heal by scar formation → Corneal opacity → LOV (Loss of Vision)

Corneal opacities :

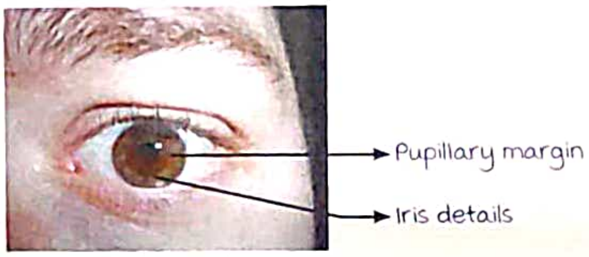
Opacity	Pupillary margin behind Opacity (macroscopic)	Iris details behind opacity (microscopic)	Distribution of visible light
Nebular (faint)	Visible	Visible	maximum  mild opacity Diffraction ↑↑ Distortion of image
Macular	Visible	Not visible	-
Leukomatous (most dense)	Not visible	Not visible	minimum  Dense opacity Light rays → don't cross opacity ↑↑ loss of vision

----- Active space -----

3. Stroma :

- Thickest corneal layer (90% of corneal thickness)

Composition : • Type I collagen fibres
• Glycosaminoglycans (GAGs) : Keratan Sulfate (m/c)


Pupillary margin
Iris details

Structures behind the cornea

4. Dua's layer / Pre-Descemet's membrane :

Strongest corneal layer

5. Descemet's membrane :

- Secreted continuously by underlying endothelial cells → Can regenerate

Schwalbe's line : Peripheral termination (at limbus) near anterior position of trabecular meshwork.

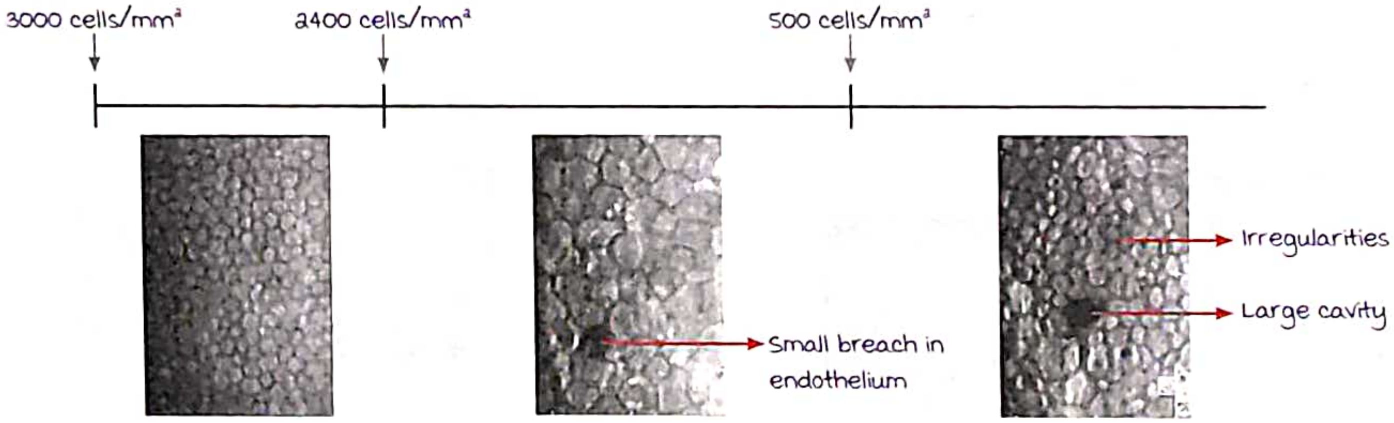
6. Posterior (Innermost) layer Endothelium :

- most metabolically active layer
- Endothelium with zona occludens → prevents entry of aqueous into cornea → maintains transparency

↓

If injured/breached → Edema of cornea → Haze/opacity.

Specular microscopy : Findings depending upon cell count.


3000 cells/mm²
2400 cells/mm²
500 cells/mm²
Small breach in endothelium
Irregularities
Large cavity

Normal endothelium
- Hexagonal cells
- Normal borders

Corneal transparency maintained

Cornea compensates by:
- Polymegathism (↑ size)
- Polymorphism (Change in shape)

↓

maintain integrity of tight junction

↓

Corneal transparency maintained

Corneal decompensation d/t extended contact lens wear : Hazy cornea

----- Active space -----

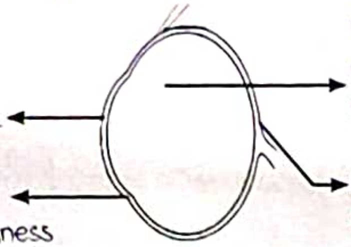
Sclera

00:52:00

Outermost covering of posterior 5/6th of eyeball

ANATOMY

Colour : White : d/t Irregular arrangement of collagen fibres.


Cornea
Limbus
(Scleral thickness 0.83 mm)
Variable thickness of Sclera
Posterior to insertion of recti :
Sclera → Thinnest
Around the optic nerve :
Sclera → Thickest.

Layers

Episclera :
- Outermost layer
- Highly vascular: 2 plexuses
  - Superficial
  - Deep : near sclera proper
- merges with Tenon's capsule : around 3 mm posterior to limbus.

Sclera propria :
- Avascular
- Collagen fibres

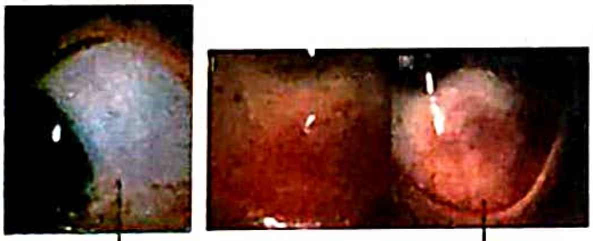
Lamina fusca :
- Innermost layer.

PATHOLOGIES

Scleral pathologies

1. Scleritis (misnomer) :

- Inflammation of the Deep vascular plexus of the episclera



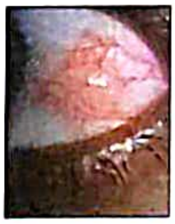
Bluish/violaceous hue (in severe cases)

- Redness remains : inflamed deep vessels do not constrict in response to vasoconstrictors

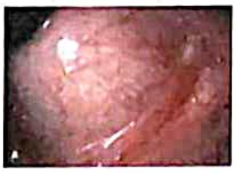
2. Episcleritis :

- Inflammation of the Superficial vascular plexus

Cause : Idiopathic (m/c)



Reddish hue

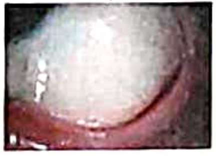


Before test

Vasoconstrictor test

Principle : pharmacological reversal of inflammation induced vasodilation

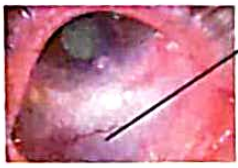
Reagent : 2.5% phenylephrine eye drops



After test : Redness disappears blanching occurs (superficial vessels constrict)

- management :
  - Self resolving, resolves fast.
  - Supportive : NSAIDs, low dose steroids.

- Types of Scleritis
  - Non necrotizing
  - Necrotizing
    - With signs of inflammation
    - without inflammation
      Scleromalacia perforans
      - m/c in women with long-standing Rheumatoid Arthritis



Scleromalacia perforans

Atrophy d/t inflammation
- Only deep (bluish) vessels seen
- No pain
- No congestion

Limbus

01:07:50

Corneo-scleral junction that contains stem cells

Limbal Stem Cells/pleuripotent cells (LSC) :
- Ability to differentiate (e.g., into conjunctival, corneal, scleral cells, etc.)
- Universal marker of stem cell : CD34.
- Specific marker of limbal stem cell : ABCG2.

Site : Limbal basal epithelial cells forming Palisades of Vogt (elevations & crypts).

Deficiency of LSC : Pterygium
- Caused by environmental factors
- Rx: Excision and autograft with limbal stem cells

----- Active space -----

ANATOMY OF UVEA, ACCOMODATION WITH ITS ANOMALIES

Components of uvea :

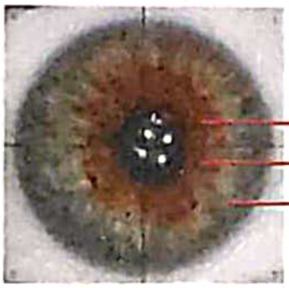
		Part of urea	Inflammatory condition
Iris		Anterior uvea	Anterior uveitis
Ciliary body	Pars plicata		
	Pars plana	Intermediate uvea	Intermediate uveitis
Choroid		Posterior uvea	Posterior uveitis

Iris

00:01:50

ANATOMY

- ↑ Vascular.
- Pigmented (d/t melaocytes).
- Thickness : Variable.



Iris

a. Thickest part:

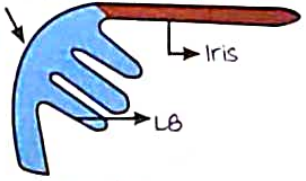
Collarette: Boundary b/w

Pupillary zone of iris

Ciliary zone of iris

b. Thinnest part :

Root : Site of iridodyalisis (detachment of iris)



Root of iris

Muscles of Iris :

	Sphincter pupillar	Dilator pupillae
Function	miosis (constriction of pupil)	mydriasis (dilatation of pupil)
Autonomic Nerve Supply	Parasympathetic control (inhibitory) ↓ via Oculomotor nerve	Sympathetic (excitatory) control Hypothalamus ↓ 1° Spinal cord : Short ciliospinal budge (C8-T1)
Final nerve supply	↓ Infraorbital branch (to inferior oblique muscle) ↓ Short ciliary nerve (Post ganglion)	Crossing lung parenchyma ↓ 2° ⟶ Resection of Pancoast tumor : Risk of damage to SCG fibres ↓ Loss of sympathetic supply to eye Superior (SCG) Cercial ganglion Along internal carotid ↓ 3° Long ciliary nerve

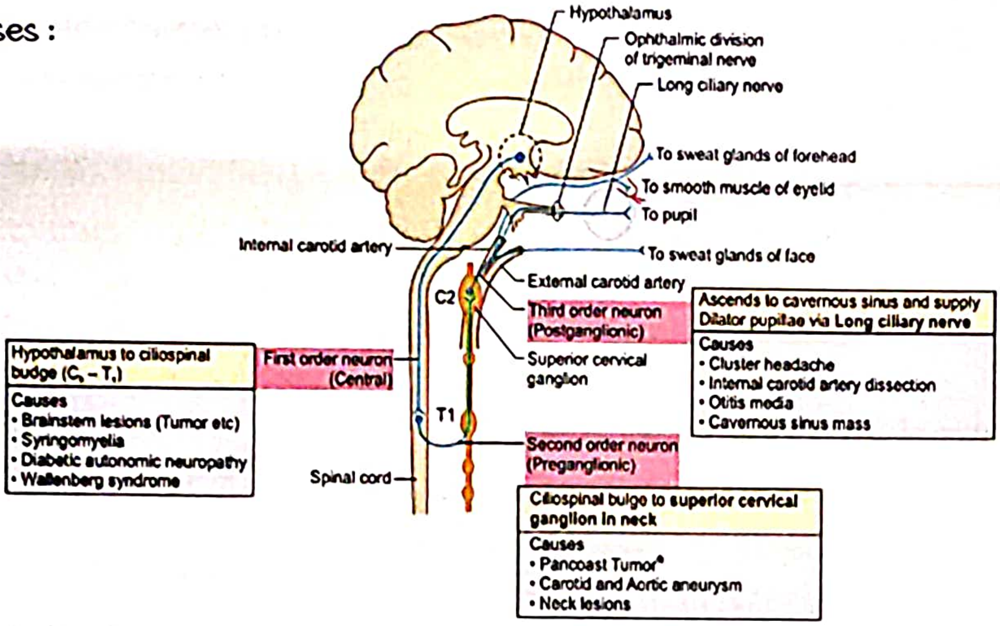
Note :

- Parasympathetic cranial nerves : III, VII, IX, X.
- Oculorator nerve
  - → Superior branch : Supplies
    - Superior rectus
    - Levoter palpebrae superioris
  - → Inferior branch : Supplies
    - Inferior rectus
    - Inferior oblique

HORNER'S SINDROME (HS)

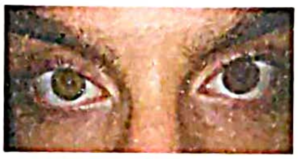
Oculosympathetic palsy (paralysis of sympathetic supply).

Causes :


Hypothalamus
Ophthalmic division of trigeminal nerve
Long ciliary nerve
To sweat glands of forehead
To smooth muscle of eyelid
To pupil
Internal carotid artery
To sweat glands of face
External carotid artery
C2
T1
Third order neuron (Postganglionic)
Superior cervical ganglion
Ascends to cavernous sinus and supply Dilator pupilae via Long ciliary nerve
Causes
• Cluster headache
• Internal carotid artery dissection
• Otitis media
• Cavernous sinus mass
Hypothalamus to ciliospinal budge ($C_8 - T_1$)
Causes
• Brainstem lesions (Tumor etc)
• Syringomyelia
• Diabetic autonomic neuropathy
• Wallenberg syndrome
First order neuron (Central)
Spinal cord
Second order neuron (Preganglionic)
Ciliospinal bulge to superior cervical ganglion in neck
Causes
• Pancoast Tumor*
• Carotid and Aortic aneurysm
• Neck lesions

Clinical features :

Maemonic : HI MAPLE



Heterochromia

could be

(R) eye hypochromic (Horner's confirmed clinically)

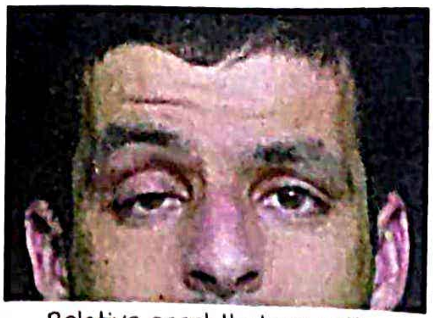
(L) eye hyperchromic

- Heterochromia Iridis (HI) :
  I/L hypochromia.
  - Only in Congenital Horner's .
- Inferior eyelid elevation : Paralysis of inferior tarsal muscle.
  (Analogous to muller's muscle in upper eyelid).
- Miosis : Paralysis of dilator pupillae → constriction occurs.
- Anhydrosis : Absence of I/L sweating.
- Ptosis : Drooping of upper eyelid : D/t paralysis of muller's muscle.
- Loss of cilio spinal reflex
- Relative enophthalmos (ptosis + inferior eyelid elevation → sunken appearance of eyeball → not true exophthalmos).

----- Active space -----

Note :

- mullers muscle / superior tarsal muscle. } Elevation of eyelid
- LPS (suppled by CN III).



Relative enophthalmos with right eye ptosis

Diagnosis :

1. Detection of Horner's Syndrome

If cocaine test :

blocks reuptake of norepinephrine (NE)

↑ NE in the cleft → Dilatation of pupil ↓ Normal pupil

No NE in cleft → No dilatation (NO NE released) ↓ Horner's syndrome

1% Apraclonidine Test (α agonist) :

- Normal selectivity : $\alpha 2$ (miosis) > $\alpha 1$
- Horner's : Denervation hypersensitivity of α_1 receptors (mushrooming)

↓

Selectivity $\alpha_1 > \alpha_2$

↓

Dilatation of Pupil

2. Localisation of lesion : 1% amphetamine test

Acts by displacement : Hydroxyamphetamine (HAH)

↓

Promotes release of NE into post synaptic cleft

Dilatation of pupil → Central pathway affected - Central HS OR Preganglionic pathway affected - Preganglionic HS

No dilatation of pupil ↓ Post ganglionic pathway (final) affected - Post ganglionic HS

Note :

----- Active space -----

Causes of mydriasis : (ITS COMB)	Causes of miosis (HI COPS)
Internal ophthalmoplegia	H-Horner syndrome
Tonic pupil (Holmes adie pupil)	Hemorrhage (Pontine)
Sympathomimetics	I-Iridocyclitis (anterior uveitis)
Closure angle glaucoma	C-Clonidine
Oculomotor 3rd nerve palsy → pupil can not constrict	Carbolic acid
mydriatics (Atropine/homatropine/ tropicamide)	O-Organophosphates Opioid (morphine)
Belladonna Poisoning	P-Parasympathomimetic drugs (Phenothiazine/pilocarpine)
	S-Sleep
	Sedatives (Barbiturates)

Ciliary Body

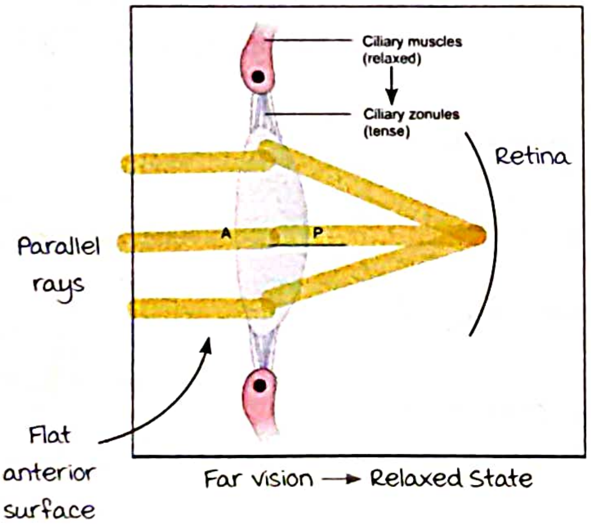
00:37:45

FUNCTIONS

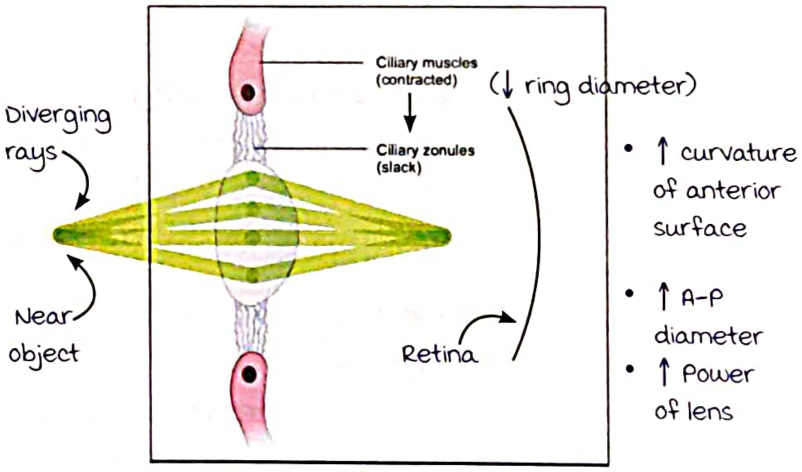
- Secretion of aqueous through ciliary processes.
- Pars plana : Site of entry into vitreous cavity (Relative avascularity → ↓ chances of bleeding).
- Accommodation :
  - Contraction of ciliary muscles → ↑ power of lens → Helps in near vision.
  - Relaxation of zonules.

Note: Components of near reflex :

a. Accommodation.
b. Convergence (inward movement of both eyes).
c. miosis.


Ciliary muscles (relaxed)
Ciliary zonules (tense)
Retina
Parallel rays
A
P
Flat anterior surface

Far vision → Relaxed State


Ciliary muscles (contracted)
(↓ ring diameter)
Ciliary zonules (slack)
Diverging rays
Near object
Retina

- ↑ curvature of anterior surface
- ↑ A-P diameter
- ↑ Power of lens

Near vision with accommodation

ANOMALIES OF ACCOMMODATION

Presbyopia :

Physiological insufficiency of accommodation → Difficulty in near vision (eg : using phones, reading newspaper).

Onset : Around 40 yrs of age.

RX : Spectacles (+) → Convex (converging)

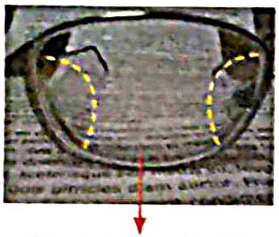
- Progressives > D-bifocals >>> executive lenses.
- Power depend on age.
  a) At 40 yrs → +1 D required.
  b) every additional 5 yrs → + 0.5 D added to power.

Types of Spectacles

for patients only needing near vision lenses

↓

monofocal



No power in peripheries → Restricted field of view

Power restricted to centre hourglass configuration → Restricted field of view

for patients already using glasses for far vision

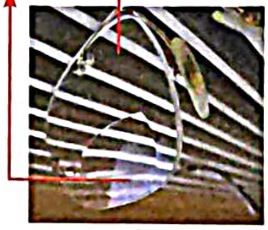
Progressive lenses

Advantages:

- Cosmetically appealing d/t seamless transition of lens
- Covers near, intermediate and far vision

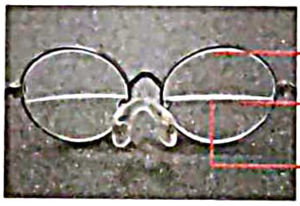
Bifocals

1. D-Bifocals
- Disadvantages
  - Cosmetically un appealing
  - No provision for intermediate vision



D Bifocals

2. Executive bifocals



Obsolete in most cases except in pediatric aphakia.

Spasm of accommodation :

continuous state of contraction of ciliary muscles

↓

loss of far vision (pseudomyopia)

Treatment : Cycloplegia (Relaxation of ciliary muscles)

- Drugs → mydriatics & cycloplegics :
  - Atropine, Homatropine, Cyclopentolate.
  - Homatropine.
  - Cyclopentolate.

ANATOMY OF RETINA

Retina - Innermost Layer of Eye

00:00:20

Neurosensory layer.

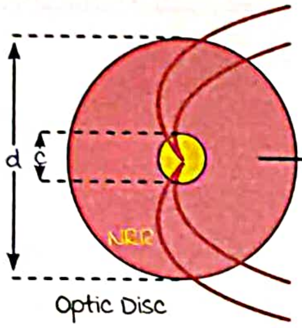
GROSS ANATOMY

1. Optic Disc (OD) :

- AKA Blind Spot of marriot (Rods & cones absent).
- All layers of retina terminate at OD (except nerve fibers).
- 1st part / Only visible part / Intra-ocular part of optic nerve (ON).
- Point in the retina where ON leaves eyeball.

Appearance :

- Shape : Vertically oval
- Diameter : 1.5mm / 1500µm
- Normal c/d ratio : → 0.3



d(OD) = C(optic cup) + NRR (Neuro retinal rim)

↓

- Allows vessels to travel through

NRR thickness

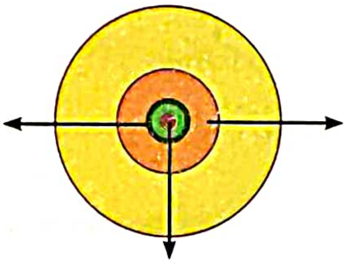
Inferior > Superior > Nasal > Temporal

↓

Visibly thinner in glaucoma

2. Macula Lutea :

- Yellowish spot
- Temporal to OD
- Diameter: 5.5mm



Foveola :
Does not have ganglionic layer

Fovea : Diameter 1.85 mm
↓
Highest/best visual acuity : maximum concentration of cones
↓
Central vision

Umbo :
Small central depression
↓ Light shone upon eye
Reflects light & shines as a small bright spot : Foveolar reflex

3. Ora Serrata (OS) :

- Anterior termination of retina
- Indicates junction between ciliary body and choroid.

Significance : Helps determine site of intra-vitreal injection.

OCULAR ROUTES OF DRUG ADMINISTRATION, BLOOD SUPPLY OF EYE AND EMBRYOLOGY OF EYE

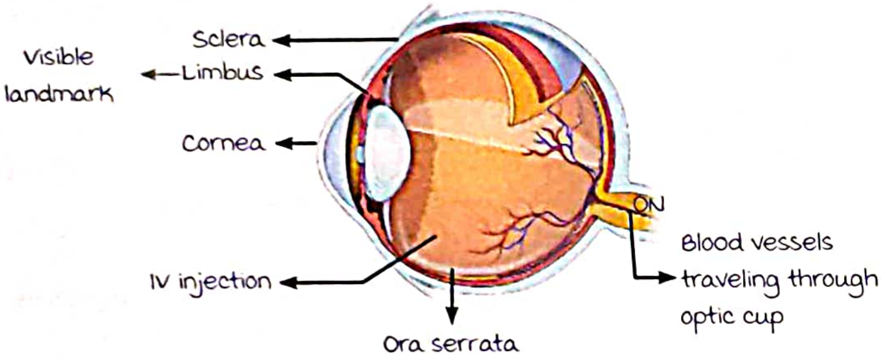
Routes of Ocular Drug Administration 00:00:15

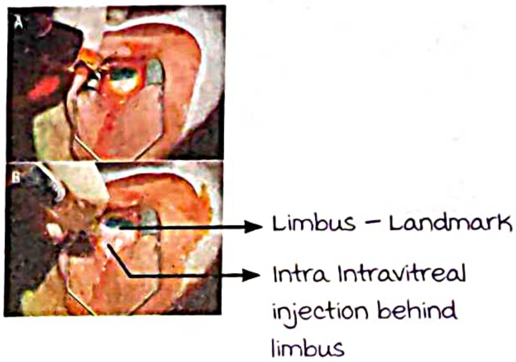
I. Intravitreal injection :

Site :

Condition of Lens	Site of intra vitreal injection
Aphakia (absence of lens)	3 mm posterior to limbus
Pseudophakia	3.5 mm posterior to limbus
Phakia (normal crystalline lens)	4 mm posterior to limbus

- Pierces : Sclera and pars plana


Visible landmark
Sclera
Limbus
Cornea
IV injection
Ora serrata
ON
Blood vessels traveling through optic cup


A
B
Limbus – Landmark
Intra Intravitreal injection behind limbus

Procedure : Using speculum, open eyes
↓
Sterilize sclera infero-temporally
↓
Ask patient to look upwards/inwards
↓
mark site of injection using caliper
↓
Injection with 30G needle is inserted through sclera with slow, twisting movements to reach vitreous.

2. Intracameral Injection :

Injection into Anterior chamber (AC)
(Eg. after incision during cataract Sx)

3. Topical :

- Application on the surface
- m/c used

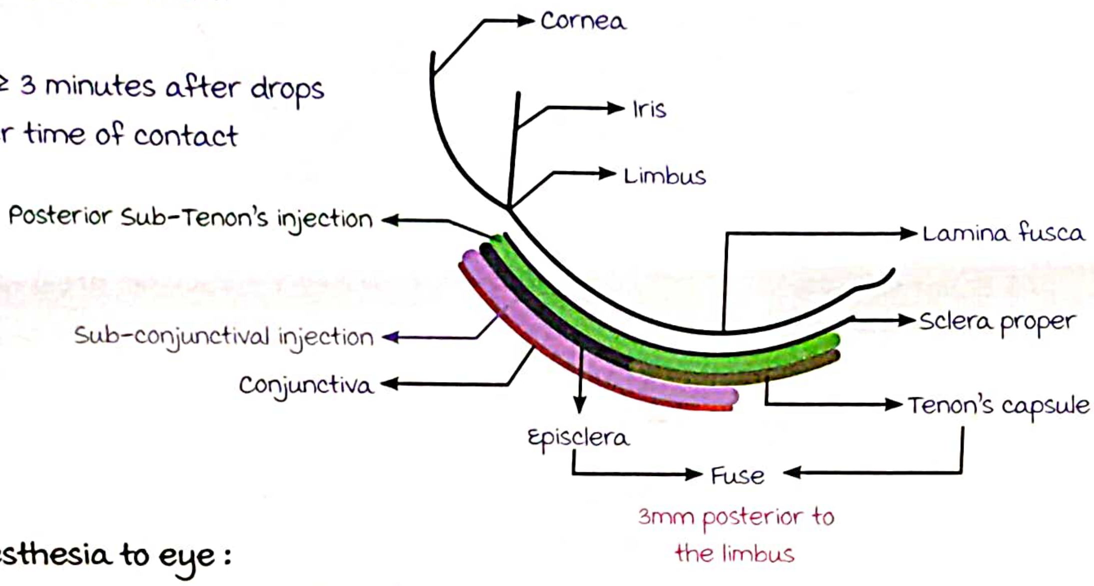
4. Subconjunctival Injection

5. Parabulbar or Posterior Subtenon (PST) Injection :

- Below Tenon's capsule
  NOZIK TECHNIQUE
- Used in Intermediate uveitis

Types :
Eyedrops : Administered before ointment
Eye ointment :

- Administered ≥ 3 minutes after drops
- Provides longer time of contact


Cornea
Iris
Limbus
Posterior Sub-Tenon's injection
Lamina fusca
Sub-conjunctival injection
Sclera proper
Conjunctiva
Tenon's capsule
Episclera
Fuse
3mm posterior to the limbus

6. Local Routes of Anaesthesia to eye :

Site : Through the lower eyelid at junction of middle 1/3 and lateral 1/3.
Procedure : Ascertain through lower eyelid below middle 1/3 and lateral 1/3.

↓

Pull lower lid down & insert injection with 24-25G needle through the lid along floor of eye

↓

Aspirate : Rule out vessel injury

↓

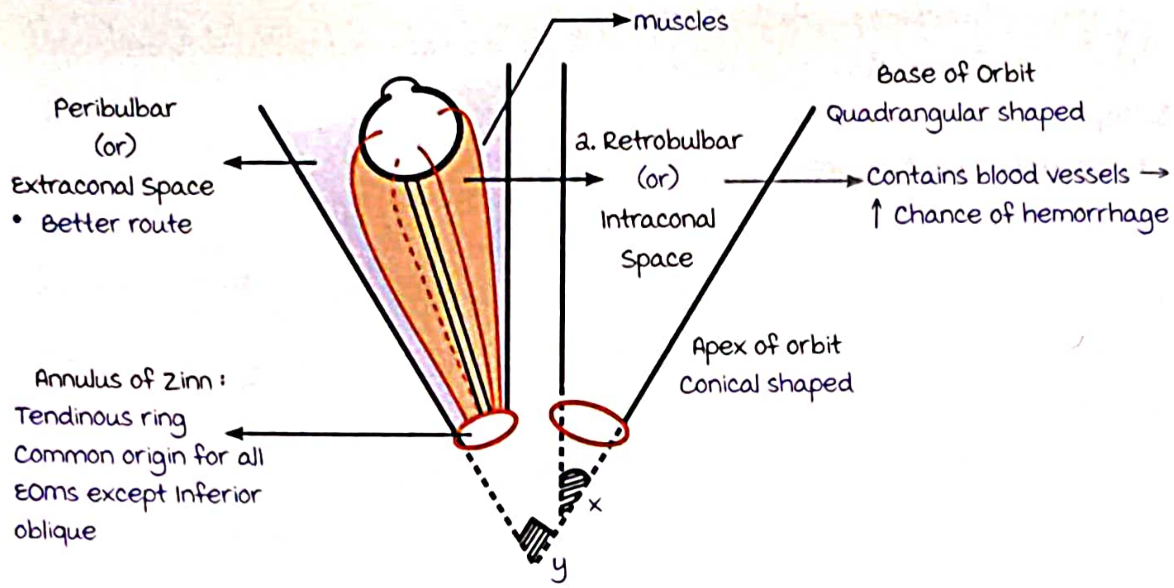
Insert 5ml of anaesthetic

↓

Post injection compression over eye to avoid rise of intra-ocular pressure

----- Active space -----

Routes of ocular anesthesia



Peribulbar
(or)
Extraconal Space
- Better route

a. Retrobulbar
(or)
Intraconal
Space

Base of Orbit
Quadrangular shaped

Contains blood vessels → ↑ Chance of hemorrhage

Apex of orbit
Conical shaped

Annulus of Zinn :
- Tendinous ring
- Common origin for all EOMs except Inferior oblique

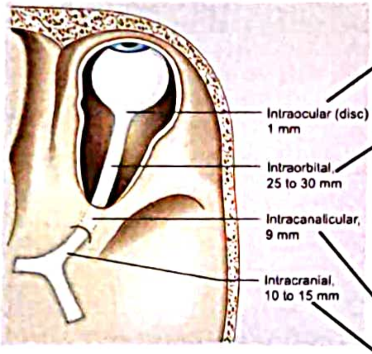
X : Angle between medial & lateral walls of orbit.
Y : Angle between both lateral walls of orbit.

Optic Nerve

00:27:50

A collection of axons of retinal ganglion cells.

Total length : 47-50 mm.


Intraocular (disc)
1 mm
Intraorbital,
25 to 30 mm
Intracanalicular,
9 mm
Intracranial,
10 to 15 mm

1st part → shortest.

2nd part → longest
- Acquires myelin sheath
- Central retinal artery & vein enter 10 mm behind the eye) inferomedially.

3rd part : within optic canal

4th part

Blood Supply of Eye

00:36:35

----- Active space -----

Internal carotid artery

↓

1. Ophthalmic artery

2. Central retinal artery
- Supplies inner 6 layers of retina
- Enters ON

3. Posterior Ciliary Artery

4. Long posterior ciliary artery
- Pierces sclera away from ON

↓

Travels b/w sclera & choroid

↓

Reaches ciliary body

↓

Supplies ciliary muscle

↓

Travels anteriorly to supply iris

major arterial circle
- Supplies ciliary process of iris

minor arterial circle
- Supplies pupillary zone of iris

5. Short posterior ciliary artery

↓

Pierce sclera around ON

↓

Supplies
- Sclera
- Choroid
- Outer layer of retina

7, 8. muscular branches to recti
- 7 in number
  - 2 for SR
  - 2 for mR
  - 2 for IR
  - 1 for LR

medial rectos

forms

6. Anterior ciliary artery

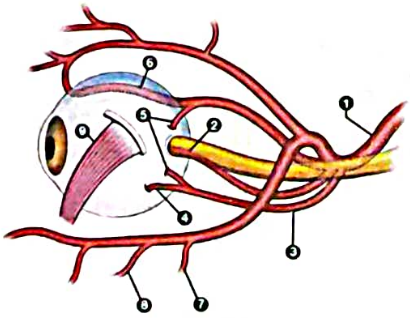
↓

Pierce sclera anteriorly, posterior to limbus

↓

Supplies :
- Anterior portion of sclera
- Ciliary process
- Iris
- Conjunctiva

SR : Superior rectus
IR : Inferior rectus
mR : medial rectus
LR : Lateral rectus



Blood supply

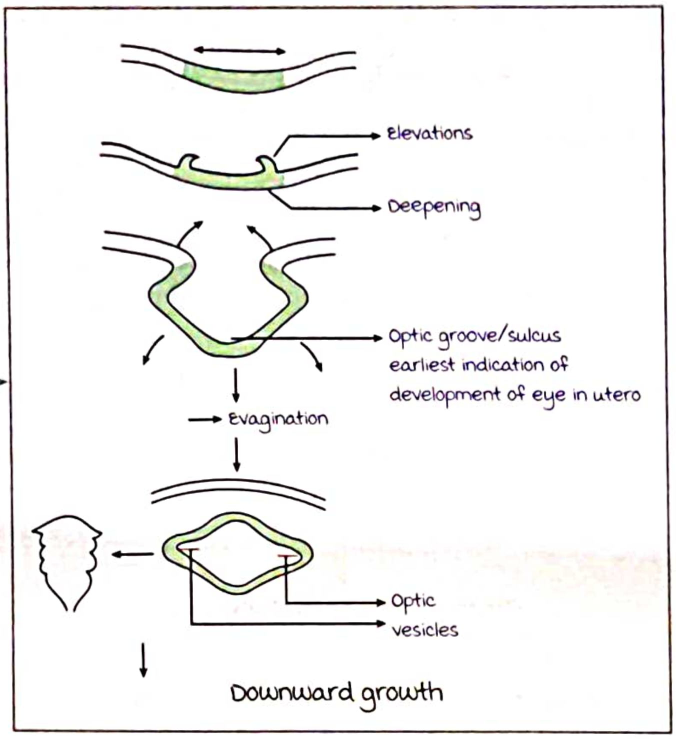
----- Active space -----

Development of the Eye

00:42:52

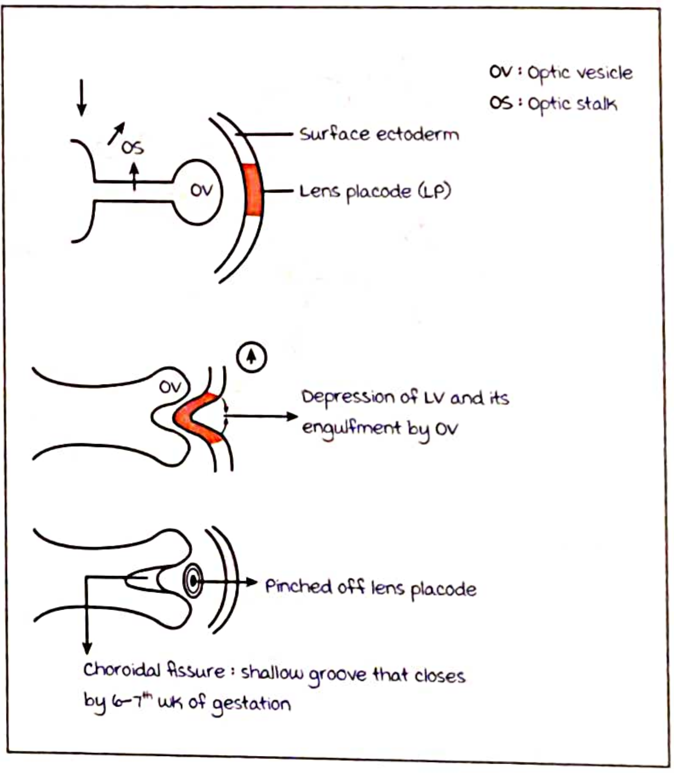
Begins on Day 22 / End of 3rd week of gestation.

Downward growth

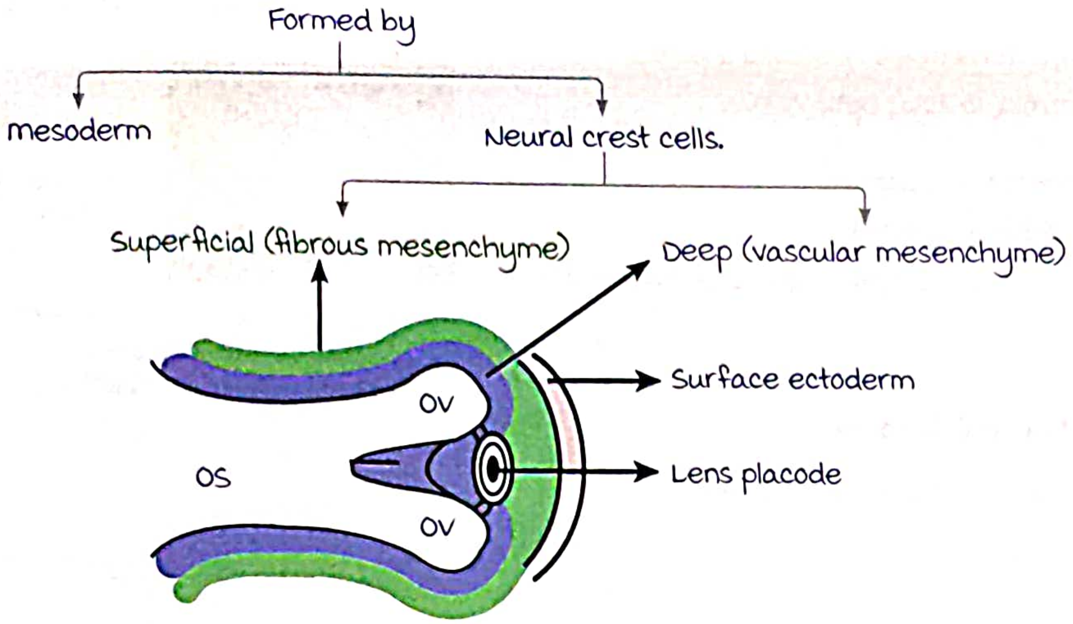

Elevations
Deepening
Optic groove/sulcus
earliest indication of
development of eye in utero
Evagination
Optic
vesicles
Downward growth

Day 27 Onwards

Lateral growth & lens formation


OV : Optic vesicle
OS : Optic stalk
OS
OV
Surface ectoderm
Lens placode (LP)
OV
Depression of LV and its
engulfment by OV
Pinched off lens placode

Choroidal fissure : shallow groove that closes by 6–7th wk of gestation mesenchyme of the eye :


Formed by
mesoderm
Neural crest cells.
Superficial (fibrous mesenchyme)
Deep (vascular mesenchyme)
Surface ectoderm
Lens placode
OV
OS
OV

Derivatives :

1. From Surface ectoderm :
   - Lens.
   - Skin of eyelids.
   - Epithelium of cornea.
   - Epithelium of conjunctiva.
   - Tarsal glands / meibomian glands.
2. From Neural ectoderm :
   - Optic nerve.
   - Retina.
   - Vitreous.
   - Epithelium of iris + ciliary body (CB).
   - muscles of iris :
     a) Sphincter pupillae (SP).
     b) Dilator pupillae (DP).
3. From Neural crest cells :
   - Uvea (vascular layer).
   - Sclera.
   - Descemet membrane of cornea.
   - Ciliary muscle.
4. mesoderm forms :
   - Extra Ocular muscles.
   - Endothelial lining of blood vessels.

Coloboma

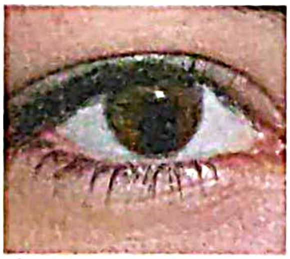
00:56:13

Etymology : missing, mutilated part.

TYPES :

1. Eye ball Coloboma

- Congenital malformation.
- d/t non-closure of choroidal fissure.



Infero-nasal iris coloboma

- Pear-shaped pupil/key hole pupil

----- Active space -----

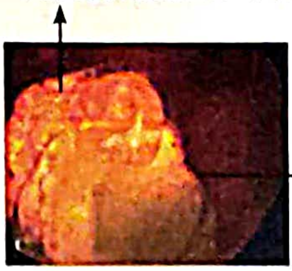
Structures affected :

a. Deep mesenchyme derivatives : Iris Ciliary body).

b. From OV & OS: Choroid, Retina, Optic nerve.

Types :
- Typical : Infero-nasal quadrant.
- Atypical : Any other quadrant.

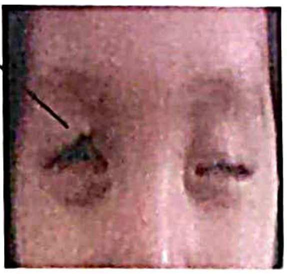
Infero-nasal iris coloboma (pear-shaped pupil) (Key hole pupil).



Retino - Choroidal / Fundal coloboma :
- ↑ Incidence of retinal detachment
- Types: Ida mann classification

2. Eyelid Coloboma :

- Do not occur due to choroidal/fetal fissure non-closure.
- Incomplete form of facial cleft/Cryptophthalmos.
- Associated with :
  - Fraser syndrome
  - Goldenhar syndrome
  - Treacher Collins syndrome



Eyelid coloboma

Complications $\xrightarrow{\text{Prevented by}}$ Rx : Eyelid reconstruction
- Cosmetic blemish.
- Exposure Keratopathy.
- Amblyopia (lazy eye) d/t development of :
  - Keratopathy.
  - Refractory errors.
  - Dermoid (Goldenhar syndrome).

3. Lens Coloboma :

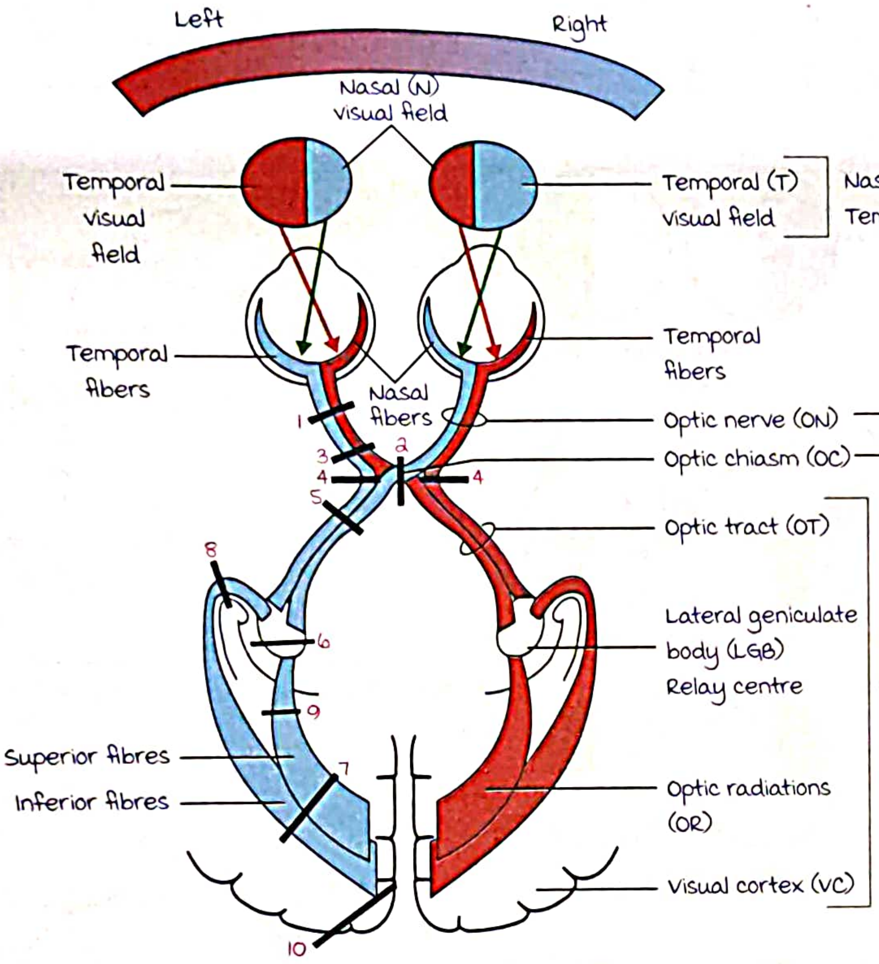
- Not a true coloboma (Lens placode : large → cannot go through choroidal fissure).
- Defective development of zonules → Notch shaped inferior defect in lens.

VISUAL PATHWAY AND VISUAL FIELD DEFECTS

Visual Pathway

00:02:00

Retina : Generates visual impulses.
↓ visual pathway → If lesion present → visual field defect.
occipital cortex : Image perceived.


Left
Right
Nasal (N) visual field
Temporal visual field
Temporal (T) visual field
Temporal fibers
Nasal fibers
Temporal fibers
Optic nerve (ON)
Optic chiasm (OC)
Optic tract (OT)
Lateral geniculate body (LGB) Relay centre
Superior fibres
Inferior fibres
Optic radiations (OR)
Visual cortex (VC)
1
2
3
4
4
5
6
7
8
9
10

Nasal visual field → Temporal fibers
Temporal visual field → Nasal fibers

Optic nerve (ON) → I/L Temporal + I/L Nasal fibers

Optic chiasm (OC) → Crossing over of nasal fibers : Contains B/L nasal fibers

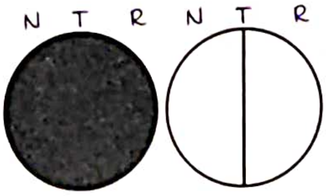
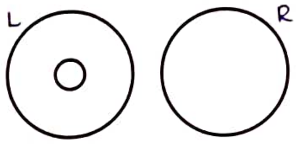
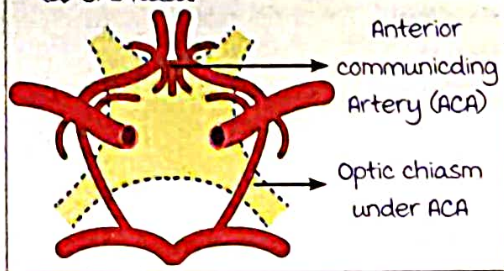
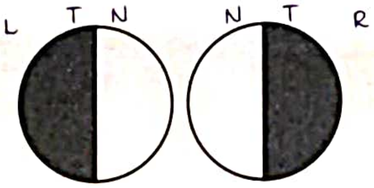
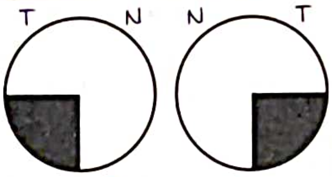
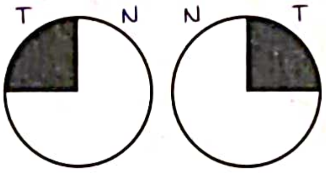
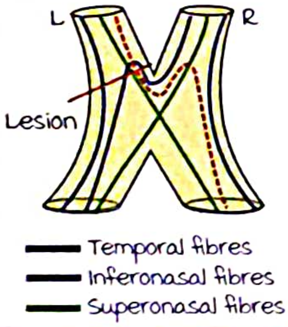
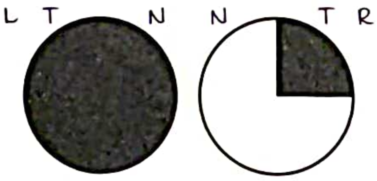
I/L Temporal & C/L Nasal fibers

----- Active space -----

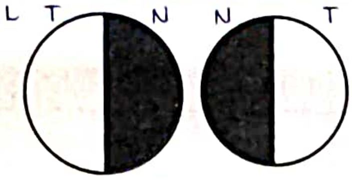
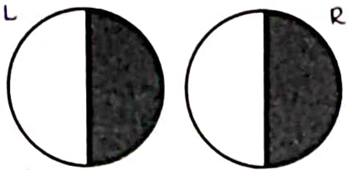
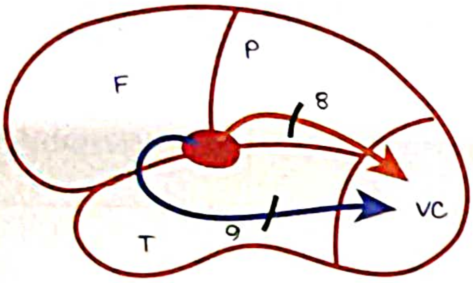
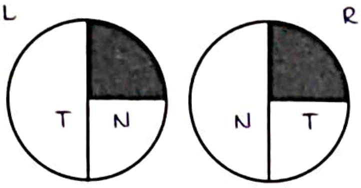
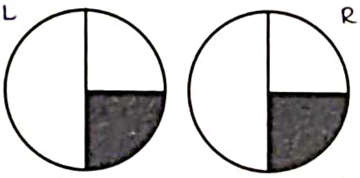
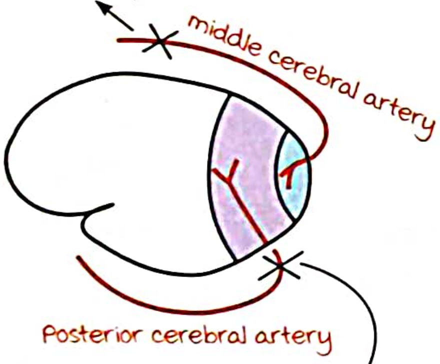
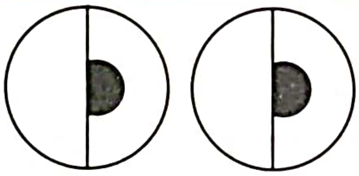
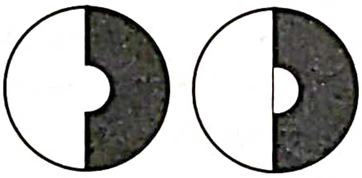
Lesions & Visual Field Defects

00:16:20

Features are described for left sided lesions.

Fibres affected	Visual field defects (VFD)	Remarks
Optic nerve : I/L Temporal + I/L Nasal	 I/L anopia (Blindness)	• macular /central fibers in ON : most susceptible to ischemia ↓ Affected first in any ON disease ↓ Earliest visual field defect : Ipsilateral central scotoma. 
2. Optic chiasma : (a) B/L Nasal  Anterior communicating Artery (ACA) Optic chiasm under ACA	 Bitemporal/Heteronymous Hemianopia	Causes : • 3rd ventricular glioma • Pituitary adenoma (from sella turcica inferiorly) • Craniopharyngioma (from Rathke's pouch superiorly) • Anterior communicating artery aneurysm • Carotid sinus thrombosis (Laterally)
(b) B/L inferonasal	 Bitemporal inferior quadrantanopia	Earliest VFD in pituitary adenoma
(c) B/L superonasal	 Bitemporal superior quadrantanopia	Earliest VFD in craniopharyngioma
3. Junction of ON & OC (Proximal ON) : Anterior Knee of Wilbrand – Lesion :  L R Lesion Temporal fibres Inferonasal fibres Superonasal fibres I/L Temporal + I/L Nasal + C/L Inferonasal	 Junctional scotoma	Cause : meningioma

----- Active space -----

4. Lateral optic chiasma : B/L Temporal	 Binasal hemianopia	very rare
5. OT 6. LGB 7. OR → I/L Temporal + C/L Nasal	 C/L homonymous hemianopsia	–
8. Superior OR : meyer's loop Superior I/L temporal + C/L nasal  9. Inferior OR : Baum's loop inferior I/L temporal + C/L nasal F : Frontal lobe T : Temporal lobe P : Parietal cortex VC : Visual cortex	 C/L : Homonymous superior quadrantanopia AKA Pie in the sky • mnemonic : TIPS (Temporal inferior pie in the sky)	• Temporal lobe disease features : – uncinate fits – Seizures – Visual hallucinations
	 C/L homonymous Inferior quadrantanopia AKA Pie on the floor	• Parietal lobe disease features : – Aphasia – Acalculia – Agraphia – Apraxia (if non dominant lobe involved)
10) Occipital lobe lesion : 10a) Tip of cortex lesion : macular representation  10b. Visual cortex except tip : I/L Temporal + C/L nasal except macular representation	 C/L homonymous macular defect AKA Cookie centre defect	• Complete occipital lobe lesion leads to C/L homonymous hemianopia and is rare
	 C/L homonymous hemianopsia with macular sparing.	

----- Active space -----

Note : Other changes seen in Lesions at 5, 6, and 7

	OT Lesion	LGB Lesion	OR Lesion
Pupil	Abnormal : Wernicke's pupil	Normal	Normal
Optic atrophy	Present bilaterally I/L : Superior & Inferior C/L : Bow-tie pattern	+	(optic radiations are post ganglionic)
Congruity of parameters of LOV	Incongruous	Incongruous	Congruous
miscellaneous	Associated with : - C/L hemiplegia - CN III palsy • Causes : - TB/Syphilitic meningitis - Posterior cerebral artery aneurysm	LGB Composed of 6 Laminae : • C/L Fibres → relay into 1,4,6 laminae • I/L Fibres → relay into 2,3,5 laminae	–

Miscellaneous Lesions

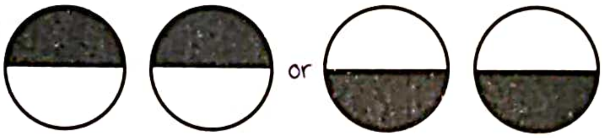
01:15:00

1. Keyhole VFD :

Cause : Incomplete LGB Lesion

2. Altitudinal VFD :

respects horizontal midline.



B/L superior B/L inferior

Causes : (mnemonic : GOA)

- Glaucoma
- Optic disc Drusen
- AION (Anterior ischemic optic neuropathy)

3. Central Scotoma :

Causes :

- Any ON (Optic Nerve) lesion:
  eg : Optic neuritis : multiple Sclerosis (m/c cause)
- Toxic optic neuropathy / tobacco-alcohol amblyopia
- Hereditary optic neuropathy
- Cone dystrophy (Stargardt's disease)

4. Cortical blindness :

Lesion in Bilateral cortical hemisphere.

Clinical features :

- B/L blindness
- Anton's syndrome : patient in denial of blindness.
- Riddoch phenomenon : Statokinetic disassociation.
- Pupil remains normal.

Diagnosis :

- VEP (visual evoked potential) : Absent
  - differentiates b/w functional & organic blindness.
  - Other uses of VEP :
    - a. malingering
    - b. Hysterical blindness

    } Normal VEP seen

PUPILLARY REFLEXES, LIGHT REFLEX AND ITS LESIONS

Pupillary reflexes :

1. Near reflex : 3 parts
   - Accomodation.
   - Convergence.
   - miosis.

2. Ciliospinal reflex : Pain in side of neck/trunk → Ipsilateral pupil dilatation.

3. Psychosensory reflex : Stress/emotion → Pupil dilatation.

4. Pupillary light reflex (PLR)

Light Reflex : Overview 00:03:24

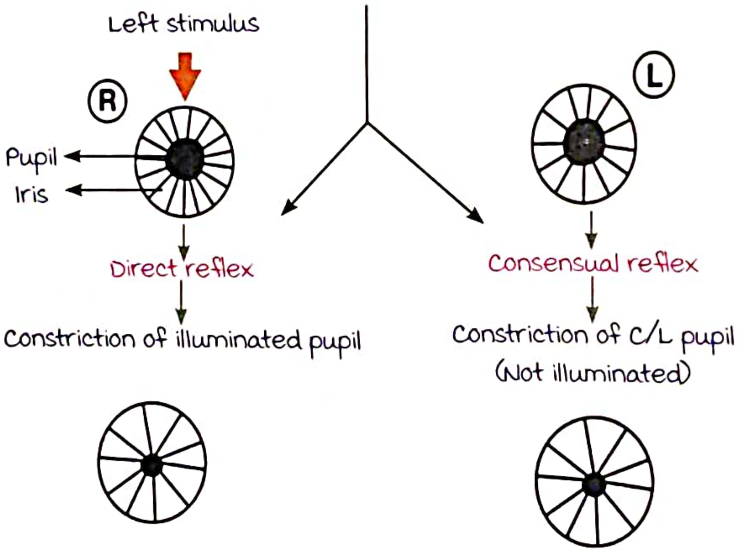
a. Procedure :

- Dark room procedure.
- Hand between eyes → Separate visual fields.

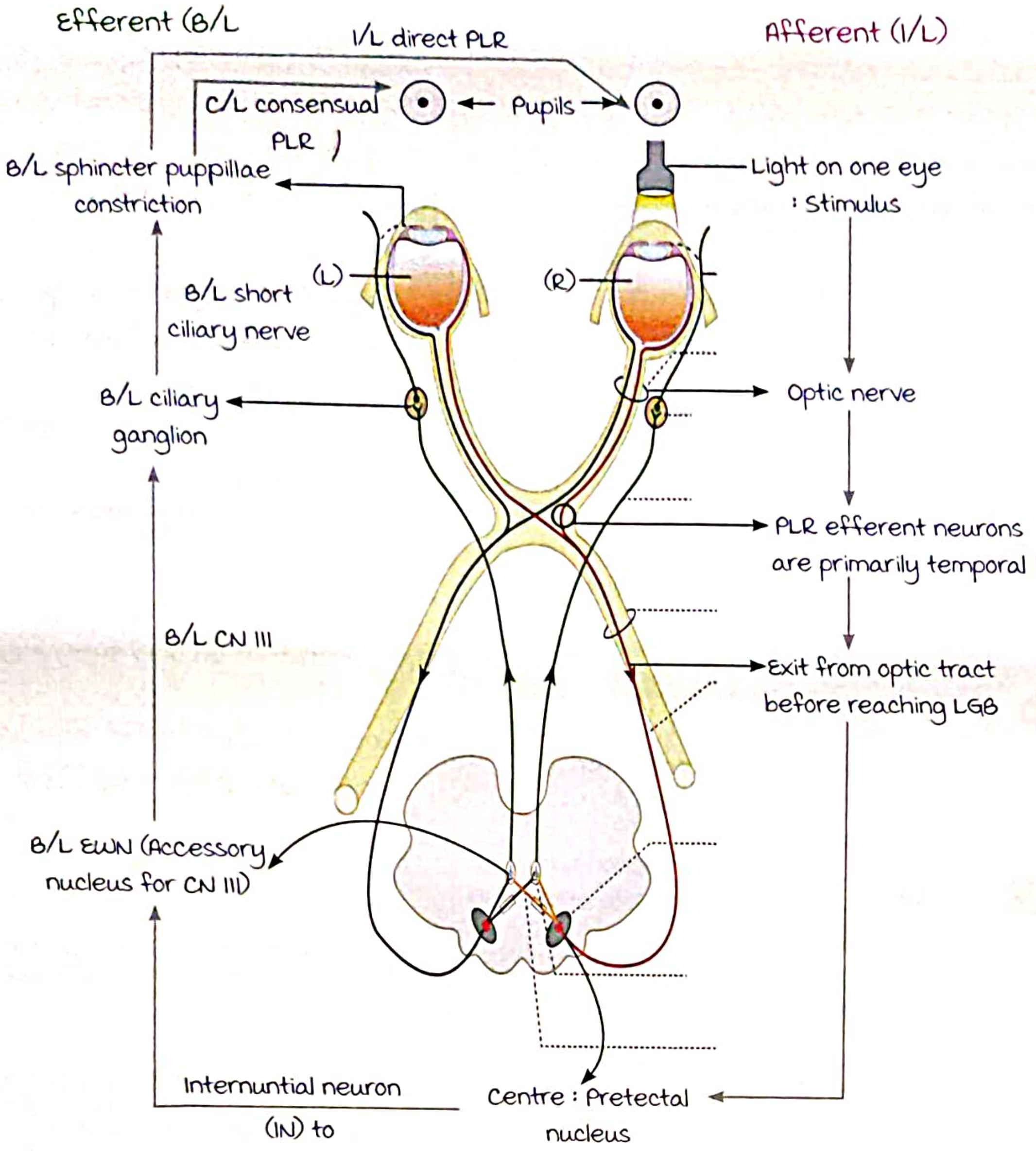
Normal findings :

- Bright light → Constriction of pupil.
- Low light → Dilatation of pupil.

b. Types :


Left stimulus
R
L
Pupil
Iris
Direct reflex
Constriction of illuminated pupil
Consensual reflex
Constriction of C/L pupil
(Not illuminated)

c. Pathway :


Efferent (B/L
I/L direct PLR
Afferent (I/L)
C/L consensual PLR)
Pupils
B/L sphincter puppillae constriction
Light on one eye : Stimulus
(L)
(R)
B/L short ciliary nerve
B/L ciliary ganglion
Optic nerve
PLR efferent neurons are primarily temporal
B/L CN III
Exit from optic tract before reaching LGB
B/L EWN (Accessory nucleus for CN III)
Internuntial neuron (IN) to
Centre : Pretectal nucleus

Note : ON, OC, OT are common to both PLR pathway and visual pathway.

Lesions of PLR

00:18:58

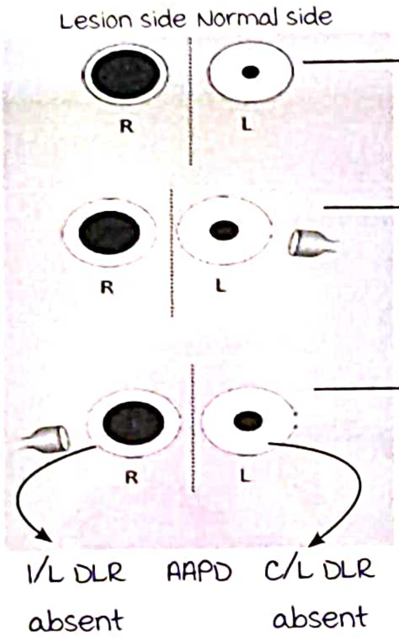
Afferent pathway lesions :

- Absolute/total afferent pathway defect (AAPD).
- Relative afferent pathway defect/marcus gunn pupil (RAPD).
- Wernicke's pupil.

Efferent pathway lesions :

- Argyll Robertson pupil (ARP).
- Tonic pupil.
- Pharmacological mydriasis.

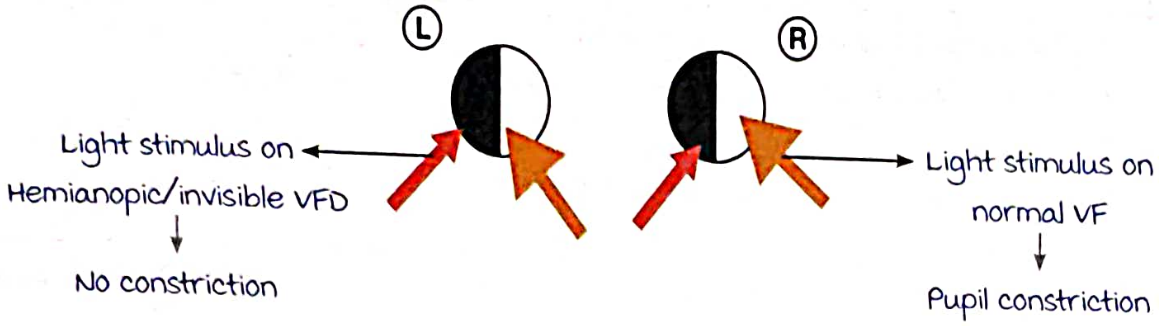
A) AFFERENT PATHWAY LESIONS

	1. AAPD	2. RAPD
Lesion	Complete optic nerve lesion d/t : • Trauma • End stage optic nerve disease	Incomplete optic nerve lesion d/t : • Optic neuritis • CRAO (Central retinal artery occlusion) • Retinal detachment (Because ON is a collection of retinal ganglion cell axons)
Findings	Light reflex test :  Lesion side Normal side R L R L R L I/L DLR absent AAPD C/L DLR absent On presentation : Light falling in both eyes → B/L pupil constricted Light on normal side (Lt eye) → B/L pupil constriction Light on lesion side → Both pupils do not constrict	Swinging flashlight test : On presentation → B/L pupils constricted ↓ Upon stimulus → B/L pupils constrict on one eye ↓ Swing flashlight to other eye as pupils dilate in the meantime ↓ Pupils constrict again ↓ Repeat : Fatigue optic nerve with lesion ↓ B/L dilation of pupils when eye with lesion is stimulated
Pupils relative to each other	Always equal in size : Isocoria	
Symptom	I/L anopia (D/t optic nerve lesion)	-

3. Wernicke's hemianopic pupil :

Site of lesion : I/L optic tract → Presentation : C/L homonymous hemianopia.

Light reflex test findings :


L
R
Light stimulus on
Hemianopic/invisible VFD
↓
No constriction
Light stimulus on
normal VF
↓
Pupil constriction

B) EFFERENT PATHWAY LESION

1. ARP :

Causes :

- Neurosyphilis : Tertiary syphilis (may present with seizures and confusion).
- Tabes dorsalis.
- Aortic regurgitation.

Site of lesion :

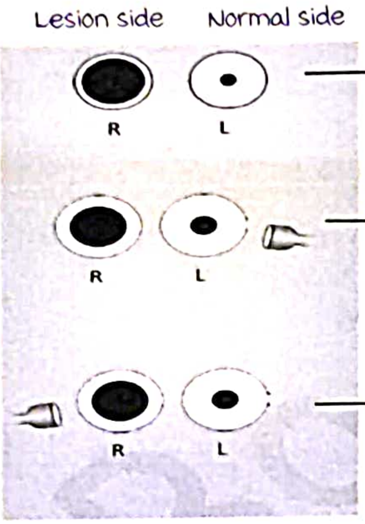
- Periaqueductal grey > Posterior commissure
  ↓ Affects ↓ Affects
  I/L internuncial neurons. C/L internuncial neurons.
- Lesions are usually B/L.

Clinical features :

- PLR : B/L absent.
- Accommodation/near reflex present.
  MOA postulate : Dorsal aspect of EWN (Containing near reflex fibres) : Normal.
- No dilation with atropine.
- On presentation : Both pupils constricted.
  - Postulate : Supra nuclear pathway destroyed → EWS receives neither excitatory/inhibitory signals
    ↓ Through ↓
    Periaqueductal area/tectum miosis
    ↓ (−)
    EWN

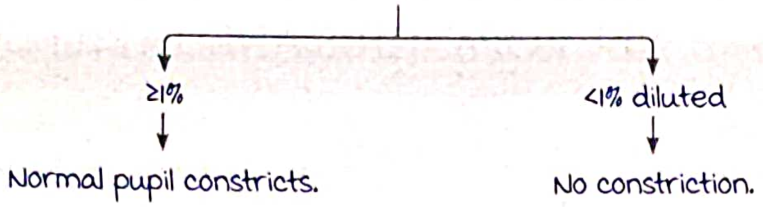
Mnemonic : ARP PRA
- Accomodation Reflex Present
- Pupillary Reflex Absent

----- Active space -----

	2. Tonic/Adie's pupil	3. Pharmacological mydriasis
Site of lesion	Ciliary ganglion/short ciliary nerve	Drug action : Supranuclear inhibition of EWN
Causes	• HZV (Herpes zoster virus) ganglionitis • Diabetes/alcoholism → Peipheral neuropathy • Trauma • Holmes Adie pupil : Tonic pupil + ↓ deep tendon reflexes	Drugs : Eg : Atropine
Ix : Light reflex	Ex : Ride side lesion/mydriatic application  On presentation Light on normal side Light on lesion side Unequal pupil size : Anisocoria → Differentiate from afferent pathway (Isocoria)	I/L pupil : Does not constrict. C/L pupil : Constricts
Clinical features	• I/L pupil DLR & CLR absent • Near reflex present : Slow, vermiform dilating movements. • Denervation hypersentivity : Constricts with 0.125% Diluted pilocarpine MOA : Sphincter pupillae denervated ↓ Overexpession of receptors (mushrooming) ↓ Hyperreaction to stimuli	Pupil does not dilate even with 1% pilocarpine

----- Active space -----

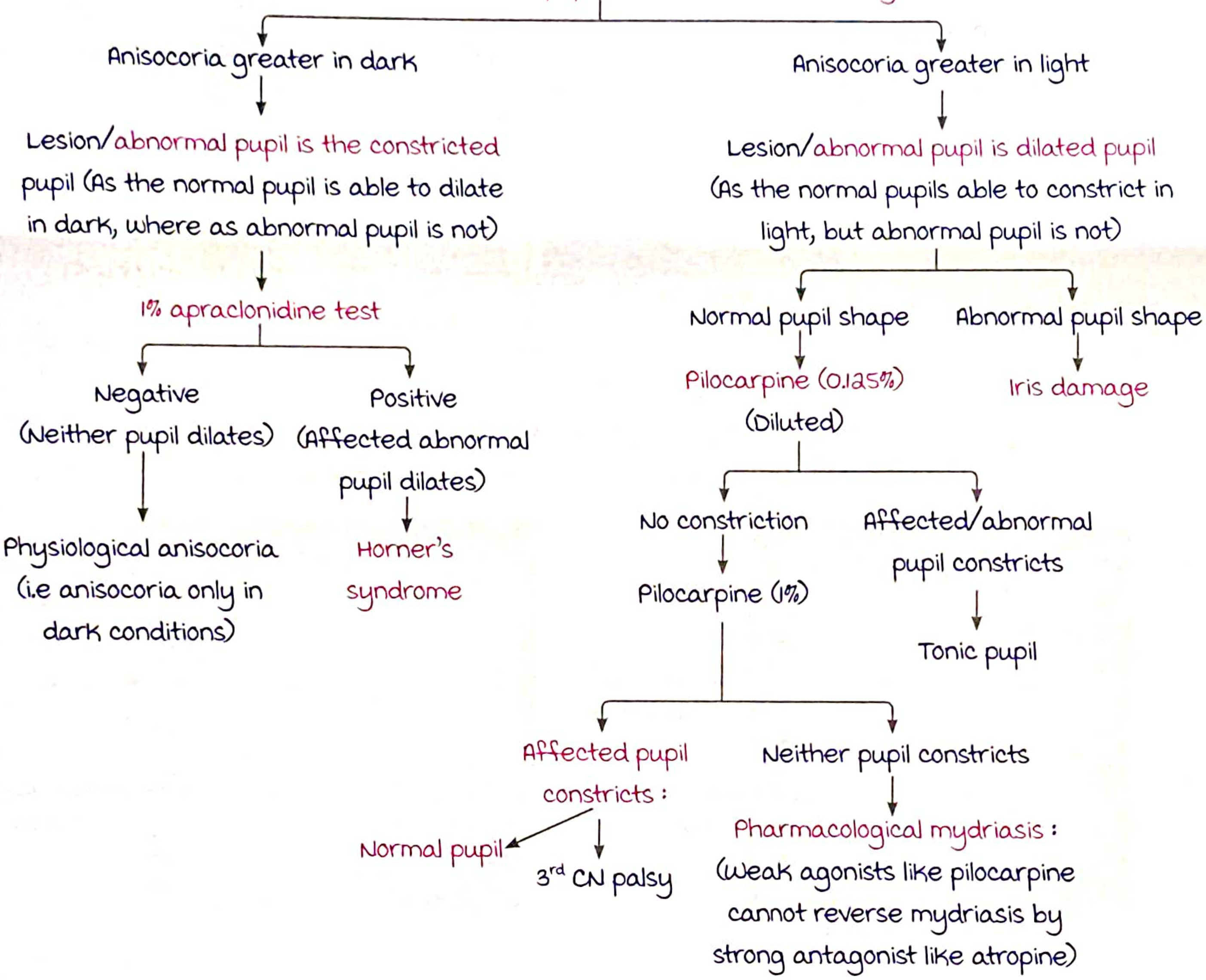
Note : Pilocarpine application on pupil



- ≥1% → Normal pupil constricts.
- <1% diluted → No constriction.

ANISOCORIA

Difference ≥0.4 mm in pupil size between the two eyes



- Anisocoria greater in dark
  - → Lesion/abnormal pupil is the constricted pupil (As the normal pupil is able to dilate in dark, where as abnormal pupil is not)
  - → 1% apraclonidine test
    - Negative (Neither pupil dilates) → Physiological anisocoria (i.e anisocoria only in dark conditions)
    - Positive (Affected abnormal pupil dilates) → Horner's syndrome
- Anisocoria greater in light
  - → Lesion/abnormal pupil is dilated pupil (As the normal pupils able to constrict in light, but abnormal pupil is not)
    - Normal pupil shape → Pilocarpine (0.125%) (Diluted)
      - No constriction → Pilocarpine (1%)
        - Affected pupil constricts : Normal pupil / 3rd CN palsy
        - Neither pupil constricts → Pharmacological mydriasis : (Weak agonists like pilocarpine cannot reverse mydriasis by strong antagonist like atropine)
      - Affected/abnormal pupil constricts → Tonic pupil
    - Abnormal pupil shape → Iris damage

OPTIC ATROPHY : OPTIC NEURITIS, OPTIC NEUROPATHIES AND PAPILLEDEMA

00:00:30

Optic Atrophy

Conditions of the optic disc is representative of the optic nerve.

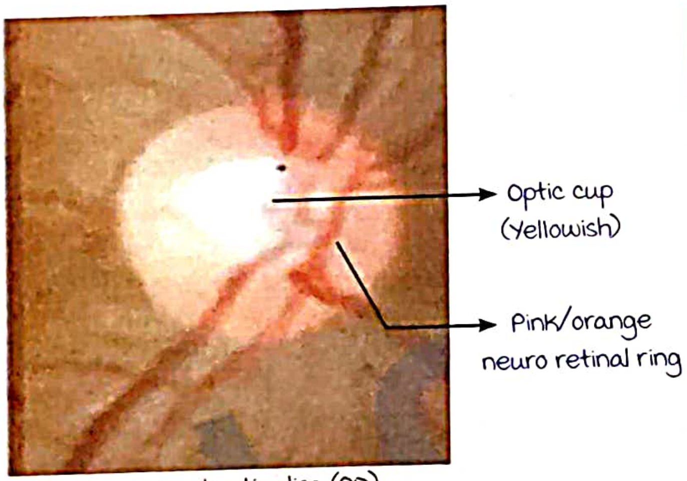
CLINICAL FEATURES

1. Pallor of OD (Optic disc) : D/t loss of vascularity.
   - First classical sign.
2. Loss of vascularity defined by Kestenbaum index : Ratio of small : largre blood vessel

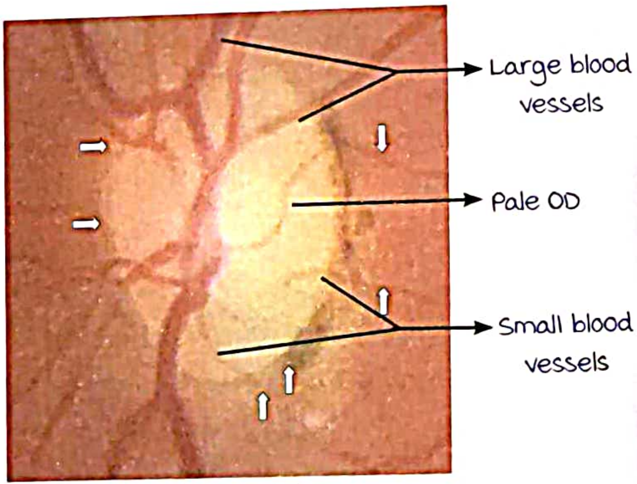
Normal ratio >10

Optic atrophy <7 → Loss of small vessels on OD

3. Visual fields loss depends on the affected fibers.
   - Patient may have 6/6 vision if central fibres are spared

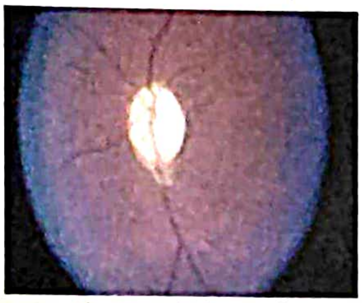
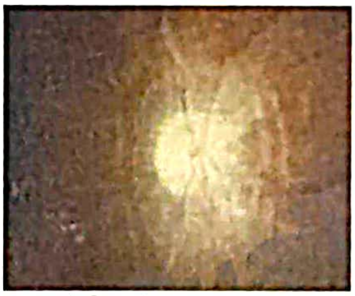

Optic cup
(Yellowish)
Pink/orange
neuro retinal ring

Normal optic disc (OD)


Large blood vessels
Pale OD
Small blood vessels

Optic atrophy

Types :

	Primary (1°)	Secondary (2°)	Consecutive
Definition	Optic atrophy without antecedent optic disc swelling	Optic atrophy after optic disc swelling	Optic atrophy due to disease of inner retina and its blood supply
Disc appearance	Chalky white, well defined margins	Dirty gray-white, ill-defined margins	Waxy pallor of disc, less sharply defined margins
Optic cup	Shallow, saucer shaped  Chalky white, well defined margins of OD	Filled up d/t swelling  Ill defined margins of OD	Normal
Vessels	Normal	Peripapillary sheathing	Attenuation
Surrounding retina	Normal	Hyaline bodies/Drusen	Pathology seen
Causes	• Optic neuritis (m/c cause : multiple sclerosis) • Tumors • Trauma • Hereditary • Toxic	• Papilledema • Papillitis • Anterior ischemic optic neuropathy (AION)	• Retinal diseases : – Retinitis seen CRAO/ CRVO – Healed vasculitis – Panretinal photocoagulation – Retinitis pigmentosa

00:15:40

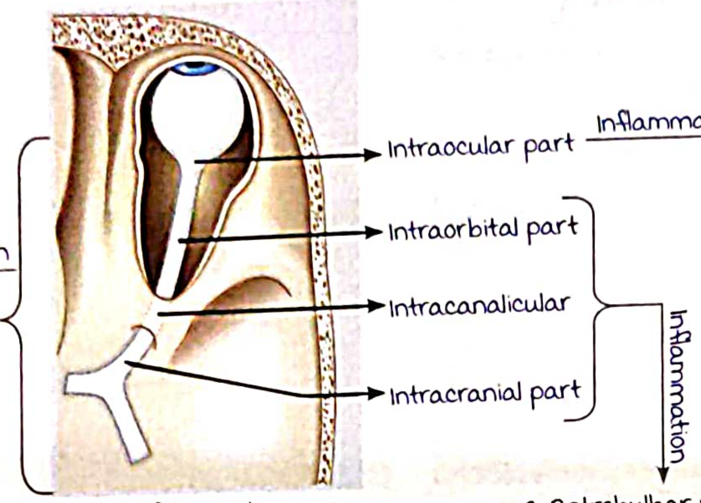
----- Active space -----

Important Causes Of 1° Optic Atrophy

OPTIC NEURITIS

Inflammation of optic nerve (ON).

Types :

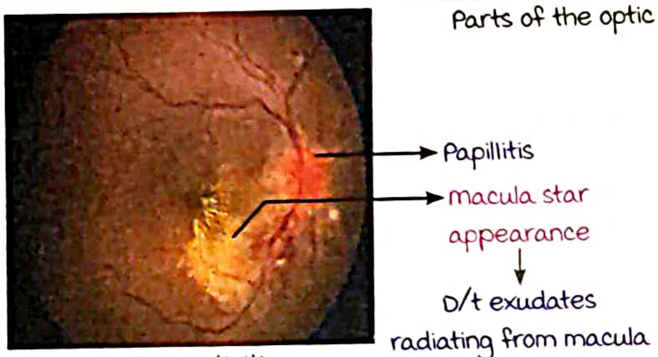

Intraocular part
Intraorbital part
Intracanalicular
Intracranial part
Inflammation
Inflammation
Inflammation

Parts of the optic nerve

1. Papillitis :
   - m/c in children.
   - Optic disc hyperemia.
   - Flame shaped haemorrhages in/ around optic disc.

2. Retrobulbar neuritis :
   - m/c in adults.
   - Clinical features :
     - No visible sign.
     - Loss of vision (+).
     - Associated with multiple sclerosis.

3. Neuroretinitis (Papillitis + maculitis)
   - macular star appearance
   - Associated with viral infections..


Papillitis
macula star appearance
D/t exudates radiating from macula

Neuro-retinitis

Note :

Causes of macular star appearance : mnemonic NHAP.

- Neuroretinitis.
- Hypertensive retinopathy.
- Angioma (Retinal).
- Papilledema.

Clinical features d/t multiple sclerosis :

mnemonic : 2 CUP mar

- Central scotoma.
- Colour blindness (Red & green).
- Unilateral (m/c) loss of vision.
- Uhthoff's phenomenon : ↑ body temperature (Eg : During exercise) → worsening of symptoms.

- Pulfrich phenomenon : Altered perception of Kinetic objects (Static → normal)

D/t : Optic nerve demyelination
↓
↓ speed of impulse conduction
↓
Inaccurate perception of fast moving objects.

- Pain in the eye : Exaggerated with ocular movements.
  - Inflammation of optic nerve → Extra ocular muscles inflammation (D/t common sheath)
    ↓
    muscles inflammed
    ↓
    Pain on movement.

- Marcus Gunn pupil/ RAPD : Earliest sign.

Treatment :

1. ONTT (Optic neuritis treatment trial) : Steriods.
   - IV methylprednisolone 1g/day for 3 days → Oral prednisone 1mg/kg/day for 11 days (Exclusive oral steriods : C/I due to ↑ chances of relapse).
   - Criteria :
     a. Vision at initial presentation : <6/12.
     b. No previous episode of multiple sclerosis and/or optic neuritis.
     c. ≥1 demyelinating lesion visible on MRI.
   - Outcome :
     a. Hastens recovery.
     b. No change in visual outcome.
2. Post steroidal β INF :
   - Concluded by CHAMPS study.
   - Good outcome → ↓ occurance of multiple sclerosis in optic neuritis patients.
3. Glatiramer acetate : 20 mg s/c injection.

NEUROMYELITIS OPTICA/DEVIC'S DISEASE

Demyelinating disease.

M/c in young females.

Cause : Can be 2° to multiple sclerosis.

Pathology : D/t aquaporin 4 (AQP-4 autoantigen).

Presentation :

- B/L optic neuritis + transverse myelitis.
- ↑ chance of relapse (more than in multiple sclerosis alone).

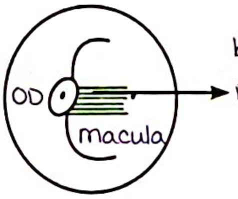
----- Active space -----

Investigations :

- measure NMO IgG antibodies (Anti AQP-4 antibodies).
- Lumbar puncture.

management : Disease control with steriods.

HEREDITARY OPTIC NEUROPATHY

Types	Genetics & inheritance	Remarks
4. KJER/Dominant optic atrophy	Autosomal dominant	-
5. BEHR	Autosomal recessive	Associations : • Ataxia • Nystagmus • mental retardation
6. WOLFRAM AKA DIDMOAD syndrome (DI, DM, Optic Atrophy, Deafness)	Autosomal recessive	Associations : • Diabetes insipidus (DI) • Diabetes mellitus (DM) • Deafness
7. LEBER's HON (LHON)	• mitocondrial inheritance ↓ maternally transmitted to m > F • Point mutation at codon 11778	Clinical features : • Presents in 2nd/3rd decade • Painless loss of vision : B/L > U/L • Pupillary telangiectasia : Dilated tortous vessels around optic disc • Pseudo-papillitis • 1° optic atrophy • Can cause central scotoma • Injury to nerve fibres between optic disc and macula : Papillo macular degeneration  Nerve fibre bundle dropof : → Damage of papillomacular nerve fibres ↓ Pathognomic feature Treatment : No treatment so far

Note :

- mitochondrial diseases do not generally have a gender predilection.
- Causes of optic disc swelling :

↑ intracranial pressure → Papilledema

Other causes (Pesudo-papilledema) :

- LHON.
- methanol poisoning.
- Near vision loss in hypermetropia.
- Optic disc drusen.
- Peripapillary myelinated nerve fibres.

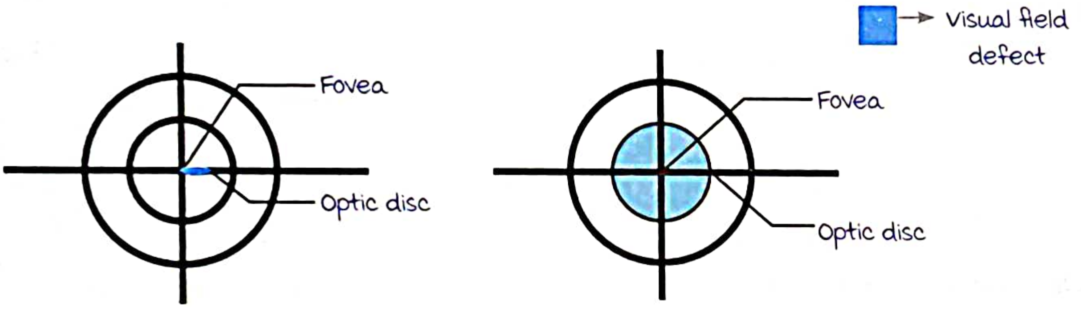
TOXIC/NUTRITIONAL OPTIC NEUROPATHY

Tobacco – alcohol amblyopia.

Causes :

- Drugs (mnemonic : DICE)
  - Digoxin.
  - Isoniazid.
  - Chloroquine.
  - Ethambutal : most ophthotoxic drug.
- Deficiency of vitamins $B_1/B_2/B_{12}$.
- methanol poisoning.

Visual field defects :



Rx : Hydroxycobalamin 1000 units weekly for 10 weeks.

Important causes of 2° Optic Atrophy 00:52:08

PAPILLEDEMA

Swelling of optic disc due to raised intra cranial pressure (ICP).

Character of swelling :

- Non-inflammatory (D/t hydrostatic pressure, passive).
- Painless.
- B/L (Except in Foster Kennedy syndrome).

----- Active space -----

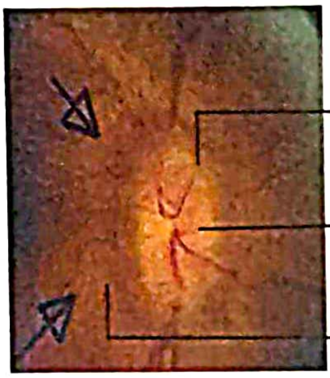
Etiopathogenesis :

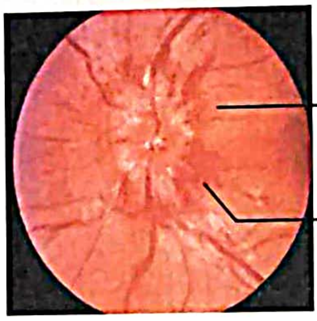
Raised ICP
↓
Pressure transmitted along subarachnoid space
↓
Pressure on optic nerve
↓
Blockage of anterograde flow/axoplasmic transport
↓
Stasis in optic disc

Extracellular edema
↓
mechanical signs

Venous congestion
↓
Vascular signs

- Acute


Blurring of disc margins (Earlier)
Elevated OD
Paton's lines : Retino choroidal folds around OD (D/t leakage of fluid)


a. Hyperemia/congestion of OD.
b. Loss of venous pulsations.
Hemorrhages around OD

- Chronic :
  Champagne cork appreance of OD.

OD swollen
↓
VFD : Enlargement of blind spot
Retrobulbar ON

Symptoms of ICP :

Visual symptoms

Papilledema :
- No LOV.
- Normal pupillary light reflex
  ↓ Leads to
  2° optic atrophy : Gradual painless loss of vison

Horizontal diplopia : D/t associated CN VI palsy

Amaurosis fugax :
- D/t brief cessation of axoplasmic transport :
  - Transient, complete LOV.
  - 30-60s episodes.
  - Total (6/6) recovery of vision post attack.
- Diagnostic of stroke.

Headache :
- Earliest sign.
- ↑ in morning.

Projectile vomiting

Progressively worsens

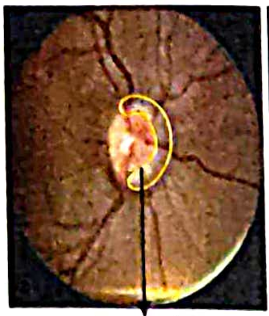
----- Active space -----

Rx :

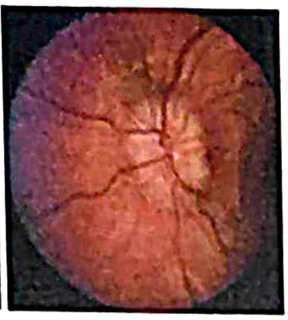
Depends on underlying cause of ↑ ICP.

Grading :

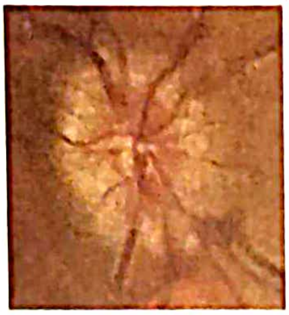
Frisen's grading for papilledema



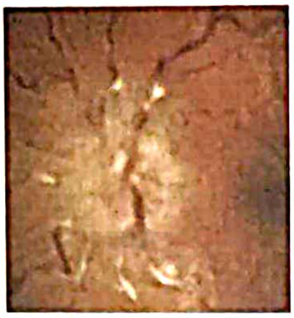
Grade 1
'C' shaped halo (Swelling) in OD



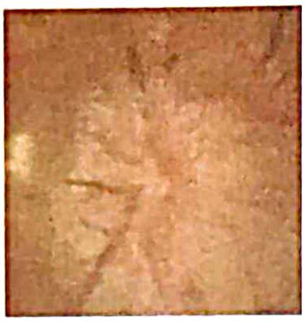
Grade 2 :
Circumferential halo
↑ swelling → Blurred OD margins



Grade 3 :
Loss of major blood vessels around disc



Grade 4 :
- Loss of blood vessels on disc
- ↑↑ swelling over disc



Grade 5 :
- All vessels obscured
- ↑↑ venous engorgement → congestion

ANTERIOR ISCHEMIC OPTIC NEUROPATHY (AION)

Loss of blood supply to anterior part of optic nerve.

Types :

	Arteritic AION cause	Non-arteritic AION (No vasculitis)
Cause	Giant cell arteritis (Large vessel arteritis)	Occlusion of short posterior ciliary artery
Associations	Temporal artery pathology	• Hypertension • Diabetes • Hyperlipidemia • Collagen vascular diseases
Symptoms	• Premonitory symptoms – Pain – Amaurosis fugax • B/L severe visual loss	• Painless • U/L moderate visual loss
Treatment	Corticosteroids	Experimental Rx : Levodopa and carbidopa for 3 weeks
Visual field defects	Altitudinal field defects	

----- Active space -----

CONGENITAL ANOMALIES OF OPTIC DISC AND COLOUR BLINDNESS

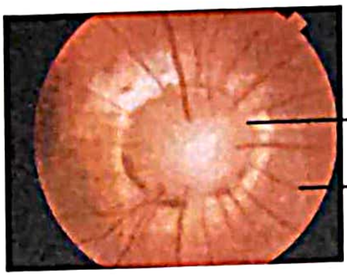
Congenital Anomalies of Optic Disc (OD)

00:00:12

MORNING GLORY SYNDROME

U/L malformation of OD.

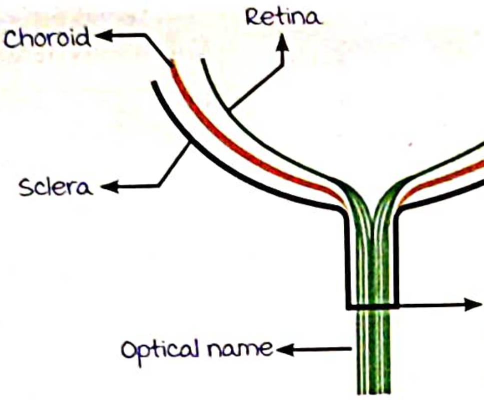
Cause : Peripapillary scleral defect d/t absence of lamina cribrosa.



→ Large OD

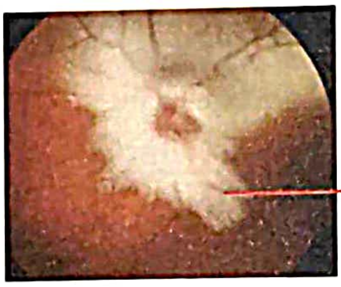
→ Radial pattern of blood vessels (Spoke wheel like)

Morning Glory Syndrome



→ Lamina Cribrosa —If absent→ Axial retrodisplacement of ON

Fenestrated lamina of sclera that allows fibres of ON to pass through



If OD acquires myelination
↓
→ White streaks around OD

MYELINATED NERVE FIBRES

- Normal OD : Non-myelinated.
- No visual loss.

COLOUR BLINDNESS (CB)

Terminology :

Primary colours :

- Red : Prota.
- Green : Deutra.
- Blue : Trita.

Pathology :

- Anopia : Non perception.
- Anomaly : Defect in perception.

Types :

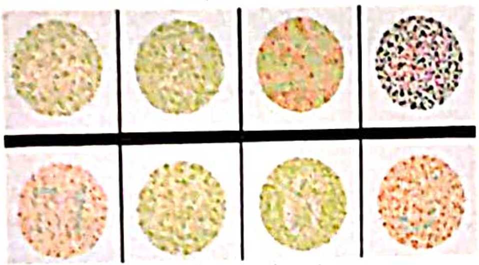
- Congenital
  - X-linked recessive inheritance (m/c) : m>F (carrier).
  - Genes affected : Red (L cones), Green (m cones) — Chromosome X
    - Blue (S cones) → Chromosome 7
  - Red & Green CB : m/c.
- Acquired

Causes	Colour affected
• Optic Nerve diseases (Eg: d/t ethambutol) - except glaucoma	Red/green CB
• macular disease - except Stargardt's disease (type of cone dystrophy without CB) • Sildenafil	Blue/yellow CB
• Old age / Digoxin	Blue CB : Yellowish tinging of vision (Xanthopsia)

Types

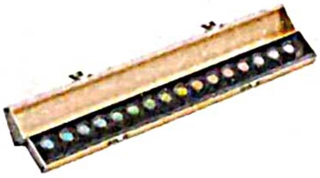
- Dyschromatopsia (defective colour vision)
  - Dichromatic
    - Anopia for 1 colour
    - Can perceive 2 other colours
    - ↓
      - a) Protanopia
      - b) Deutranopia
      - c) Tritanopia (Autosomal dominant inheritance)
  - Trichromatic anomalous
    - Can perceive all 3 colours with anomalies in perception of 1 or 2
    - ↓
      - a) Protanomaly
      - b) Deutranomaly
      - c) Tritanomaly
- Achromatopsia (absent colour vision)
  - Red monochromat
    - ↓ (Black/white)
    - Autosomal recessive inheritance
    - Total colour blindness
    - ↓ Hemeralopia (day blindness)
  - Cone monochromat
    - ↓ Perception of only 1 colour

Tests :

	Ishihara chart	Hardy Rand Ritler test
use	• m/c used to detect CB • Cannot detect tritan defects	Pediatric age group
Principle	Pseudoisochromat principle	
Charts	38 plates - Numbers - Lines → find false positives & false negatives  Ishihara chart	24 plates → shapes (O, Δ, X)

3. Holmgren's Wool test.
4. Nagel's anomaloscope : most accurate test for severity of CB.
5. Farnsworth munsell 100 hue test : • most sensitive test.
   • Can detect all types of CB.
6. Farnsworth munsell 15 hue test : Differentiates b/w congenital & acquired CB.
7. Lantern test : Vocational CB testing.

Farnsworth munsell 100 hue test



Farnsworth munsell 15 hue test

SQUINT : EXTRAOCULAR MUSCLES AND BINOCULAR SINGLE VISION

Squint : Deviation from binocular vision.

Overview :

Deviation of direction of vessels in one/both eyes & impairment in ocular movement

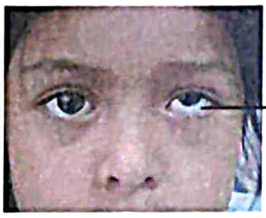
Causes :

Muscular disorders :

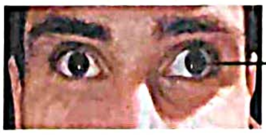
- Myasthenia gravis
- Atrophy
- Dystrophy

Cranial nerve lesions :

- CN IV $\xrightarrow{\text{supplies}}$ Superior oblique
- CN VI $\xrightarrow{\text{supplies}}$ Lateral rectus
- CN III $\xrightarrow{\text{supplies}}$ other extraocular muscles
- (mnemonic SO4LR6O3)



upward deviation of left eye



Outward deviation of left eye

Extra Ocular Muscles (EOM)

00:04:10

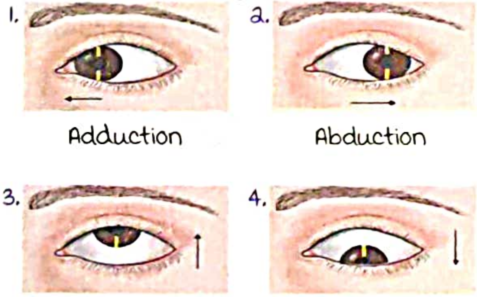
ANATOMICAL ACTIONS

- Ability to be free from neural control

  Based on direction of tendons.

Muscle	Action on single eye (ocular) Direction		
a. Levator Palpebrae Superioris (LPS)	Elevation of upper eyelid		
b. Medial Rectus (MR)	Inward movement: Adduction		
c. Lateral Rectus (LR)	Outward movement: Abduction		
d. Cyclovertical muscles	1° actions	2° actions	3° actions
1. Superior Rectus (SR)	Elevation	Intorsion	Adduction
2. Inferior Rectus (IR)	Depression	Extorsion	
3. Superior Oblique (SO)	Intorsion	Depression	Abduction
4. Inferior Oblique (IO)	Extorsion	Elevation	

Clinically visible



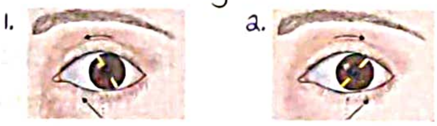
Adduction

Abduction

Elevation

Depression

Clinically invisible



Intorsion

- Inward/nasal rotation

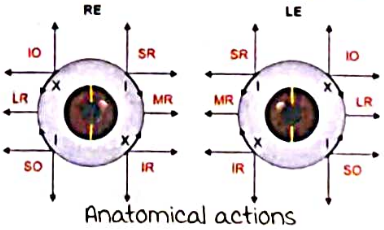
Extortion

- Outward/ temporal rotation

CLINICAL ACTIONS

Rule :
- 1° actions predominate in abducted eye.
- 2° actions predominate in adducted eye.

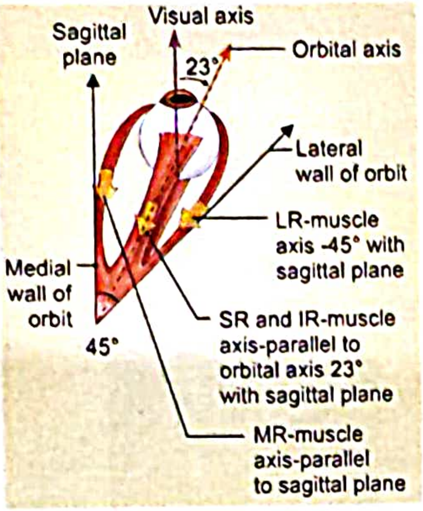
Use :
- To test actions of individual muscles.



Anatomical actions

----- Active space -----

Principle :

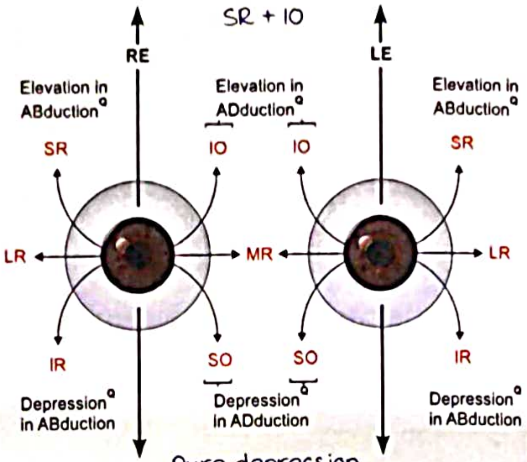

Sagittal plane
Visual axis
23°
Orbital axis
Lateral wall of orbit
LR-muscle axis -45° with sagittal plane
Medial wall of orbit
45°
SR and IR-muscle axis-parallel to orbital axis 23° with sagittal plane
MR-muscle axis-parallel to sagittal plane

Visual & orbital axes

If eye abduct by 23°
↓
Visual axis and muscle orbital axis coincide
↓
Vertical action predominate
↓
SR → pure elevator

Pure elevation

SR + IO


RE
LE
Elevation in ABduction°
Elevation in ADduction°
Elevation in ABduction°
SR
IO
IO
SR
LR
MR
MR
LR
IR
SO
SO
IR
Depression° in ABduction
Depression° in ADduction
Depression° in ABduction

Pure depression

SO + IR

Clinical functions

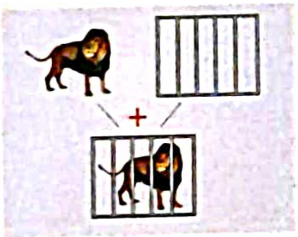
Binocular Single Vision (BSV)

00:31:00

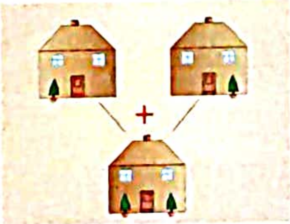
Formation of a composite image using both the eyes.

Grade

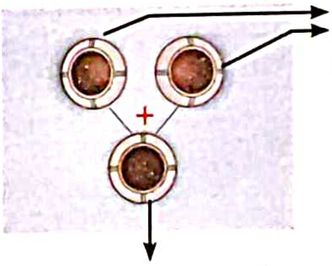
Grade 1 : Simultaneous macular perception



Grade 2 : Fusion of similar images with minute retinal differences



Grade 3 : Stereopsis binocular depth perception


Slightly varying angles

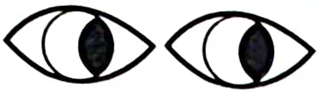
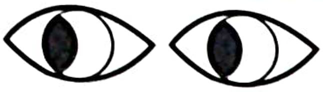
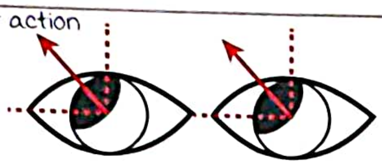
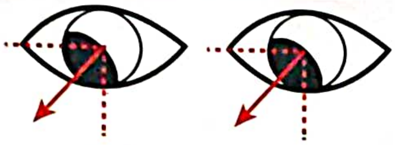
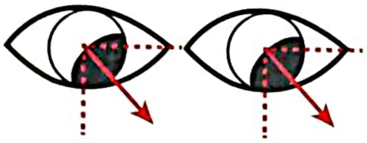
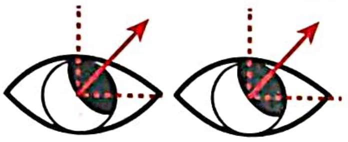
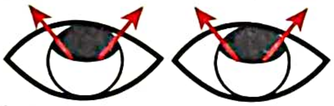
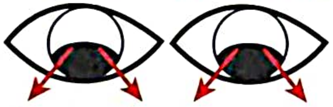
Single vision created

FORMATION OF BSV

1. Synergistic muscles :
2 muscles with the same action in the same eye :
Eg : Right SR + Right IO
(both cause elevation)

2. Antagonistic muscles :
2 muscles with opposite action in the same eye :
Eg : Right LR & Right MR
(abduction & adduction)

3. Yoke muscles / CN synergists :
A pair of muscles one from each eye enabling both eyeballs to move in the same direction : version movement.

muscle pairs	Actions
Left LR + Right MR	 Levo version
Right LR + Left MR	 Dextro version
Right SR + Left IO	Net action  Dextro-elevation
Right IR + Left SO	 Dextro – depression
Right SO + Left IR	 Levo – depression
Right IO + Left SR	 Levo – elevation
Right SR + Right IO & Left SR + Left IO	SR + IO IO + SR  B/L Elevation : Sursum version
Right IR + Right SO & Left IR + Left SO	B/L depression : Deorsum version  IR + SO SO + IR

----- Active space -----

Hering's Law of Equal Innervation

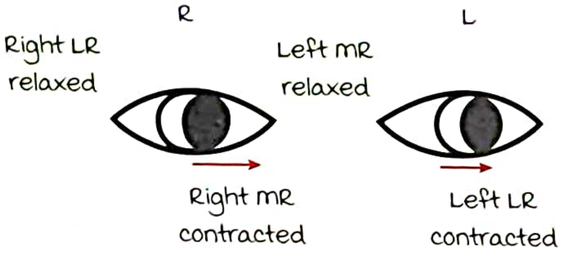
Equal innervation to a pair of yoke muscles during any binocular movement (Similar timing & amount of nerve impulses & simultaneous contraction).

Applications :
- Secondary deviation or
- Inhibitional palsy

Sherrington's Law of Reciprocal Inhibition :

Relaxation of yoke muscle antagonists.

E.g :



Formation of BSV :

4 muscles required — 2 Yoke → } Squint
2 Antagonists — Lesions → } Squint

SQUINT : CLASSIFICATIONS AND DIRECTIONS

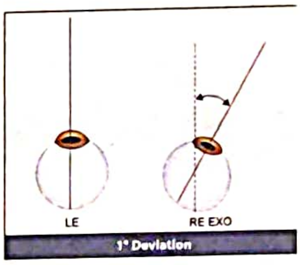
Classification of Squint

00:00:15

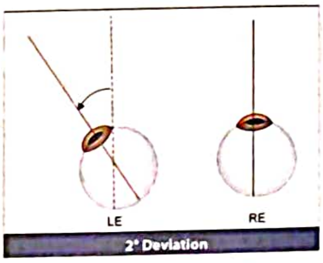
- Squint
  - Apparent deviation
Pseudostabismus
  - Tone deviation
    - Heterophoria (Latent squint) : Deviation manifests under ocular stress (Excessive near work)
    - Heterotropia (manifest squint) : Visible always
      - Constant / Concomitant Squint : Deviation remains constant in all directions of gaze
      - Incomitant Squint : Deviation varies in different directions of gaze

COMITANT & INCOMITANT SQUINT

	Comitant	Incomitant
Deviation	1° deviation = 2° deviation	1° deviation < 2° deviation
Ocular movements	Restriction absent	Restriction present
Diplopia	Absent	Present
Head posturing	Absent	Present
Sensory anomalies	Present	Absent



Deviation seen when normal eye fixates on an object



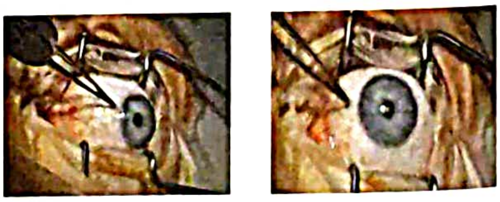
Deviation seen when deviated eye fixates on an object

Note :

Hering's law: equal and simultaneous innervation flows through yoke muscles.

Types of Incomitant Squint

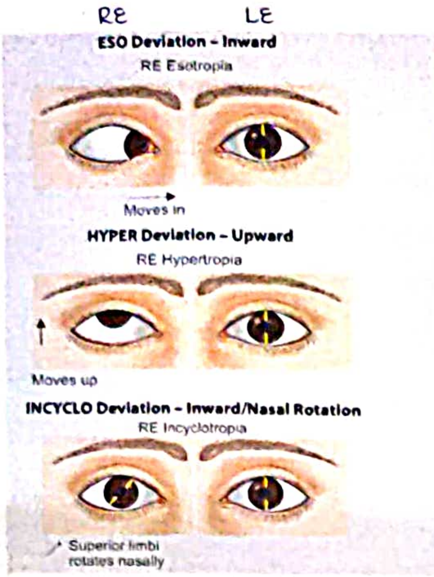
- Paralytic Squint
Forced duction test is Negative (eye moves)
- Restrictive Squint
Forced duction test is Positive : Diagnostic (eye does not move)

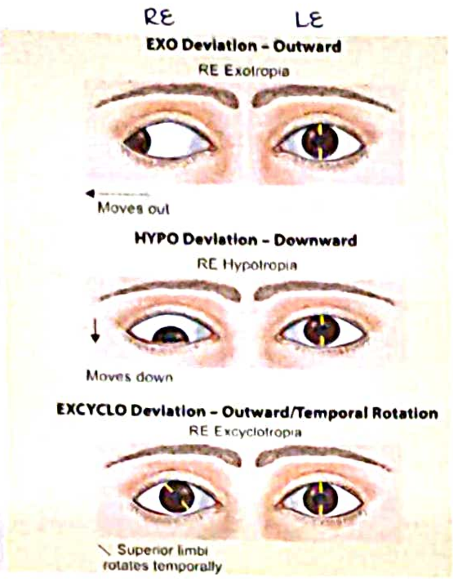


Forced duction test : Try to move eyeball being held at limbus by forceps.

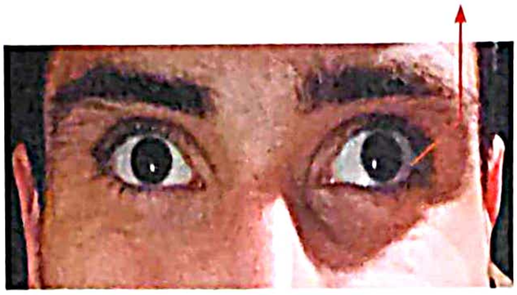
DIRECTION OF DEVIATION

----- Active space -----

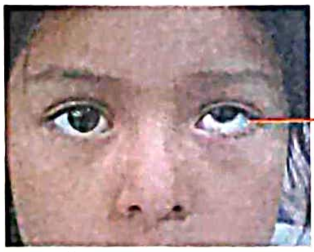

RE
LE
ESO Deviation – Inward
RE Esotropia
Moves in
HYPER Deviation – Upward
RE Hypertropia
Moves up
INCYCLO Deviation – Inward/Nasal Rotation
RE Incyclotropia
Superior limbi rotates nasally


RE
LE
EXO Deviation – Outward
RE Exotropia
Moves out
HYPO Deviation – Downward
RE Hypotropia
Moves down
EXCYCLO Deviation – Outward/Temporal Rotation
RE Excyclotropia
Superior limbi rotates temporally

Outward deviation



Left eye exotropia



upward deviation

Left eye hypertropia

Consequences of Squint

00:29:45

	Diplopia	Confusion
Seen in	Small angle squint	Large angle squint
Image formation	2 images of the same object formed at non-corresponding points in both retinae (Corresponding point of LE : nasal → temporal)	2 images of 2 different objects d/t both eyes looking at different objects
motor Adaptations	Fusion : Extra work performed by extraocular muscle to overcome small angle deviation	Change in head posture
Sensory Adaptations	• ARC : Anomalous Retinal Correspondence – Formation of new correspondence points with deviation upto 5°.	• Suppression of visual sensory impulse from the deviated eye ↓ • Amblyopia (lazy eye): Loss of vision without organic cause – no treatment

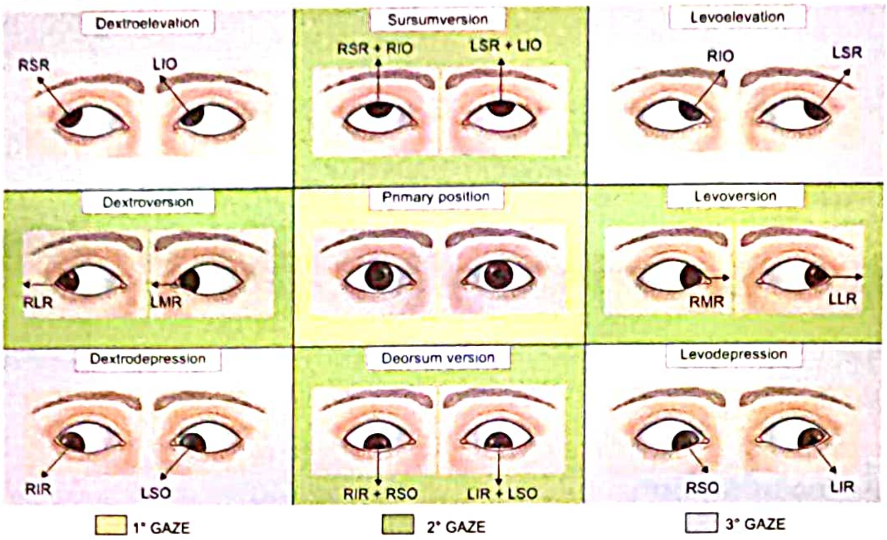
----- Active space -----

SQUINT : INVESTIGATIONS

Tests for Movements and Vision

00:00:11

EXAMINATION OF OCULAR MOVEMENTS


Dextroelevation
RSR
LIO
Sursumversion
RSR + RIO
LSR + LIO
Levoelevation
RIO
LSR
Dextroversion
RLR
LMR
Primary position
Levoversion
RMR
LLR
Dextrodepression
RIR
LSO
Deorsum version
RIR + RSO
LIR + LSO
Levodepression
RSO
LIR
1° GAZE
2° GAZE
3° GAZE

9 directions of Gaze

MEASUREMENT OF REFRACTORY ERROR

Visual acuity : Snellen's chart
- 6/6 : Normal
- <6/6 : Refractory error → measured by Retinoscope

Accommodative Squint :

Uncorrected Hypermetropia

Treatment : use of spectacles

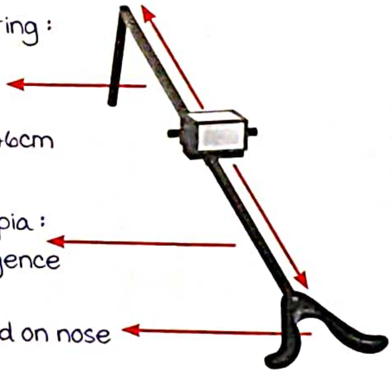
↓

Loss of near vision → Overaccomodation + Overconvergence → Inward deviation : Esotropia

MEASUREMENT OF FUSIONAL VERGENCE

Extra power of extraocular muscles to keep heterophoria from manifesting.

Tests for fusional vergence :
- a) Livingston gauge test
- b) Pencil test
- c) Synoptophore
- d) RAF (Royal Air Force) rule : m/c used - 2 points measured.



move until patient reports blurring :
- marks near point of accommodation
  - Normal 20y - 8cm, 50y > 46cm

move until patient reports diplopia :
- marks near point of convergence
  - Normal <10 cm

Placed on nose

RAF ruler

Treatment for poor fusional vergence : Orthoptic strengthening exercises.

Measurement of Ocular Deviation

00:14:12

Objective test :

1. Hirschberg's test
2. Bruckner's test
3. Cover-uncover test
4. Prism - Bar Cover

Subjective test :

Based on the ophthalmoplegic principle :

- Maddox Rod
- Maddox Wing

Based on haploscope principle :

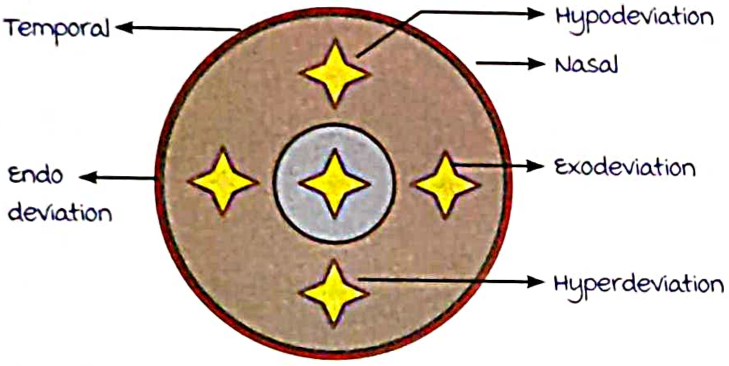
- Lancaster RG test
- Synoptophore

OBJECTIVE TEST

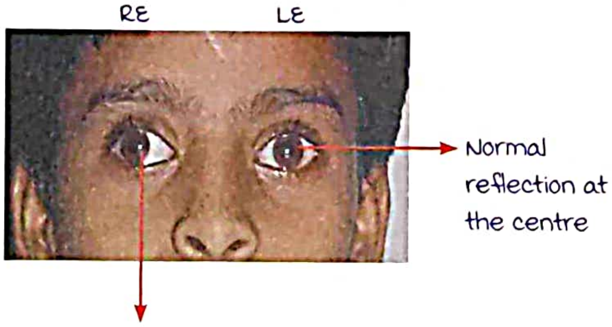
Hirschberg (corneal reflection) test :

a. Diagnosis of direction of deviation :

Mnemonic : DOOR Deviation Opposite of Reflection.


Temporal
Hypodeviation
Nasal
Endo deviation
Exodeviation
Hyperdeviation

Corneal reflection in right eye

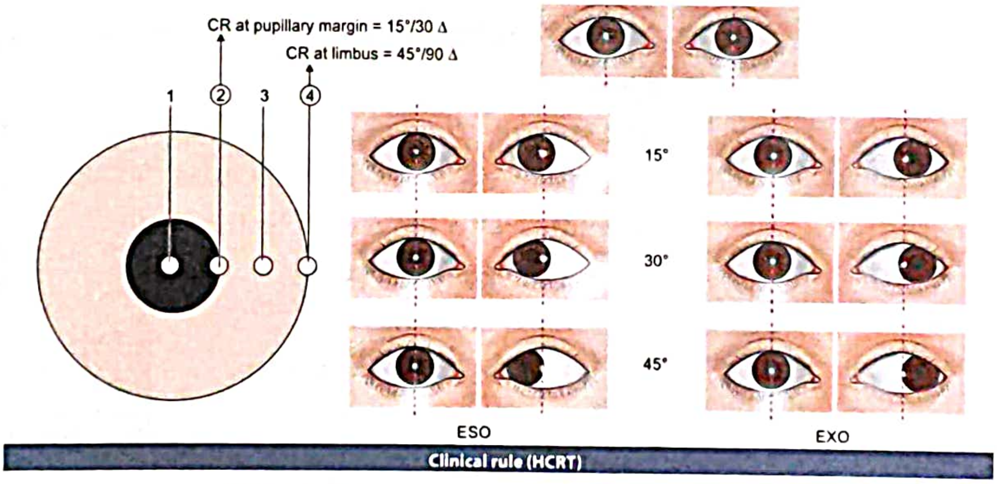

RE
LE
Normal reflection at the centre

Corneal Reflection (CR) : Nasal to centre

- 15° exodeviation (at pupillary margin)

b. Measurement of degree of deviation :

- 1mm deviation of CR from center of pupil is equal to 7° squint / 14 prism dioptre measurement : 1° = 2pd (1 pd = 1/2°).

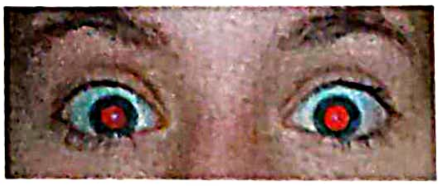

CR at pupillary margin = 15°/30 Δ
CR at limbus = 45°/90 Δ
1
②
3
④
15°
30°
45°
ESO
EXO
Clinical rule (HCRT)

Degree deviation measurement by a photograph

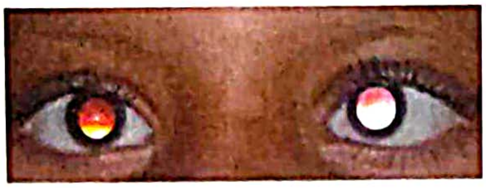
Bruckner's test :

- Screening procedure in infants.
- Shine light on eye through direct ophthalmoscope.
- Examine fundal red reflex.

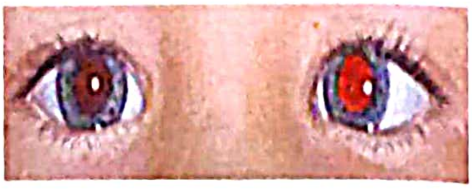
----- Active space -----



i. Red reflex : Normal
Equal b/w both eyes



ii. Arc-like reflex : Refracting error



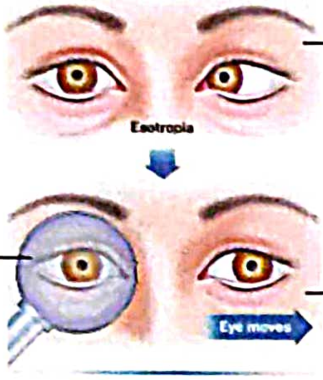
R - Darker L - Lighter
iii. Unequal Squint

Cover - Uncover test :

Cover test : Confirm Tropia Uncover test : Diagnose phoria

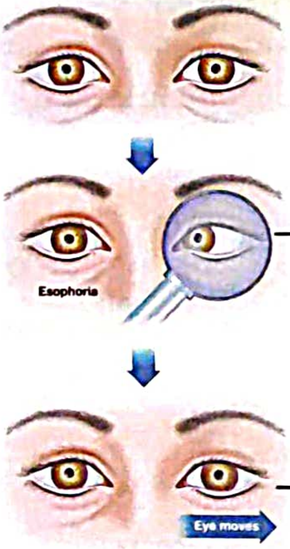
Mnemonic : DOOM, Deviation Opposite Of Movement.

i.


Esotropia
Eye moves
On presentation
Cover normal eye
Corrective inward movement
Opaque cover

Cover test

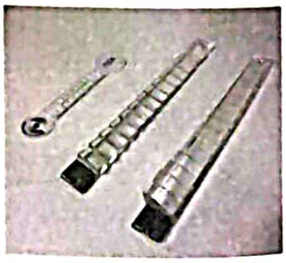
ii.


On presentation with no deviation on stress
Covers affected eye
Deviation under cover
Esophoria
Removal of cover
Movement of eye back to normal
Eye moves

Uncover test

Prism bar cover test / PBCT :

- Best & most accurate test for the measurement of degree of tropia.
- Principle : Neutralization → light passing through a prism deviates towards the apex
- Goal : Finding power of prism at which eye appears normal on cover-uncover test.
- Mnemonic : DOOB Deviation Opposite Of Base of Prism
- Orientation of prism during test:
  - ESO : Base out prism
  - EXO : Base in prism
  - Hyper : Base down prism
  - Hypo : Base up prism



Prism bar cover

SUBJECTIVE TESTS

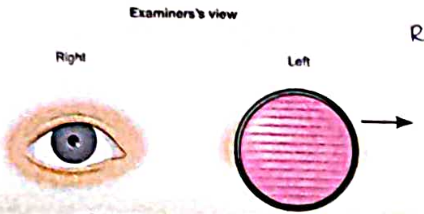
Based on Diplopia principle :

a. maddox Rod test :

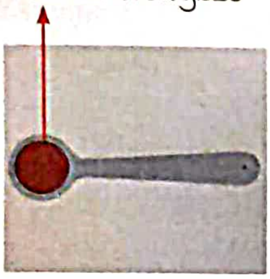
uses :

1. measure / diagnose phoria at far fixation.
2. macular function test.
3. Double maddox Rod test : measure cyclodeviation.

- method :



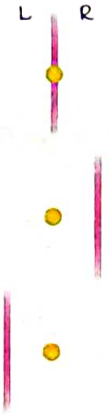
Striated red glass



maddox rod

Results :

Patient's view



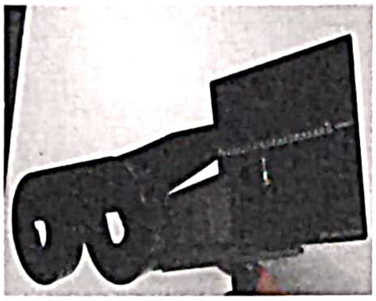
1. Orthophoria (Normal)
2. Left Exophoria /tropia (crossed diplopia)
3. Left Esophoria /tropia (urcrossed diplopia)

Note :

One test to differentiate between Tropia & Phoria : Cover-Uncover test

b. maddox wing :

- Diagnoses phoria at near fixation
- Obsolete



maddox wing

Diplopia and Tests

00:56:25

Types of Diplopia :

monocular :

- Diplopia does not disappear on closing the normal eye
- Eg : Subluxation of lens
- Severe refractory errors (Anisometropia)
- Iridodonesis

Binocular :

- Diplopia disappears if either eye is closed

Eg : Squint d/t :

- myogenic cause
- Neurogenic (3, 4, 6th nerve palsy)
- Neuromuscular junction disorders (myasthenia gravis)

Tests :

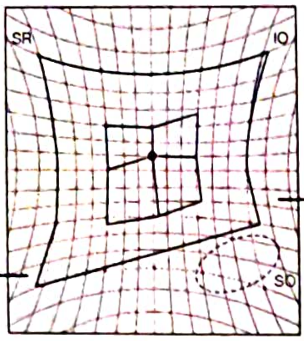
a. Red-green glasses :

Easiest way to check for diplopia

b. Lee's screen

c. Hess chart : most commonly used for Diplopia

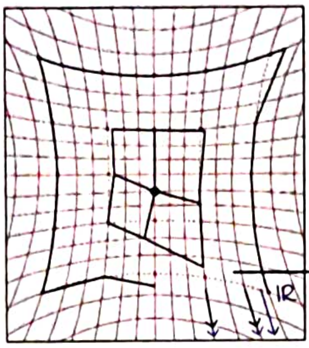
----- Active space -----



Normal Field of vision

Field of vision in depression in adduction : paralysed ISO

LE with squint



Normal RE

Compensatory field of vision by yoke muscle (right inferior rectus) in accordance with Hering's law

Note :

A chart with straight lines used in retina : - Amsler's grid

Tests for Sensory Anomalies 01:04:05

To differentiate between

1. BSV : 1 object 1 image
2. ARC : Adaptation to diplopia
3. Suppression : Adaptation to Confusion.

a. Worth's 4-dot test :

more common/more important test > Bagolini striated glasses test.

method :

Red : Worn on Right eye

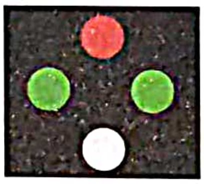
Green : Worn on Left eye

↓

Shown 4 dots.

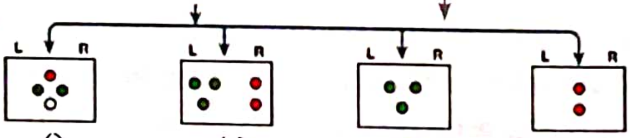
Red green glass

Patient sees



Worth's 4 dot (Normal)

Possible results :



(1)
- Normal BSV
- Harmonious ARC

(2) Diplopia
- Non Harmonious ARC

(3) RE Suppression (D/t Amblyopia)

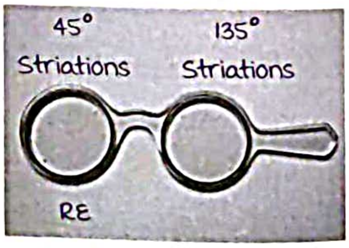
(4) LE Suppression

b. Bagolini striated glasses :

- Glasses with oblique striations.
- Shine a light → Images perpendicular to striations.

↓ Images seen individually

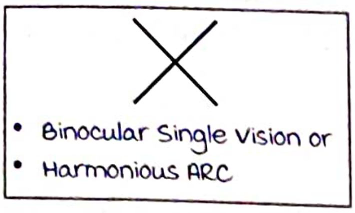
RE / \ LE



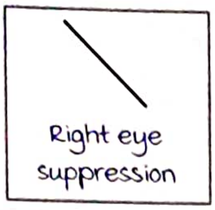
Bagolini Striated glasses

Results :

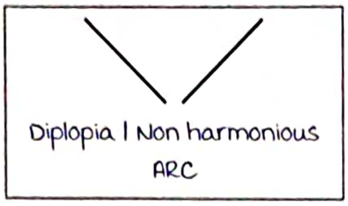
(i)


Binocular Single Vision or
Harmonious ARC

(ii)


Right eye suppression

(iii)


Diplopia / Non harmonious ARC

c. 4 Prism test :

used to differentiate between BSV & central suppression scotoma in microtropia (small angle squint).

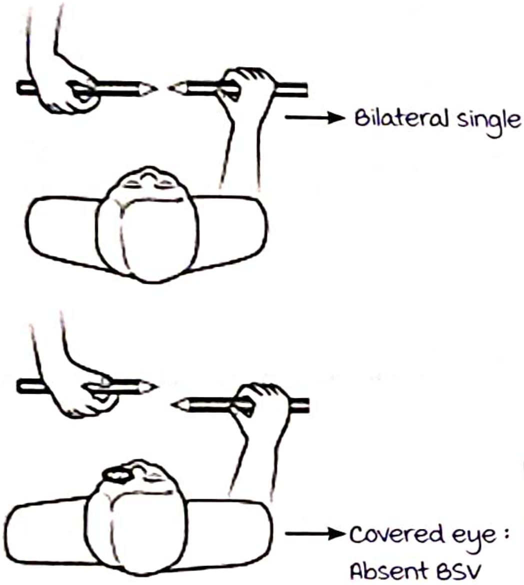
d. After Image Test.

Tests for Stereopsis (G3 BSV : Depth Perception)

01:14:00

Test for depth of perception → Integrity of BSV → Indirect test for squint

1. Two Pencil test : Gross stereopsis

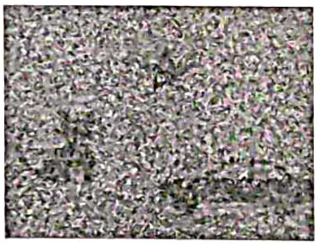

Bilateral single vision : Present
Covered eye : Absent BSV

Two pencil test

2. Titmus fly test : Polaroid vectographic images (matteawan gray)

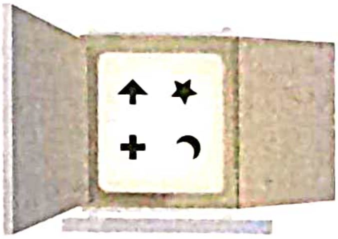
3. Random Dot Stereogram test : Best test

- to eliminate monocular clues of depth perception
- Types :



a. Lang test

b. Frisby test



c. Frisby - Davis Distance stereotest

----- Active space -----

PARALYTIC SQUINT

Type of incomitant squint

Features :

- Diplopia
- Abnormal head posture
- Restriction of ocular movements

} Intended action of paralysed muscle

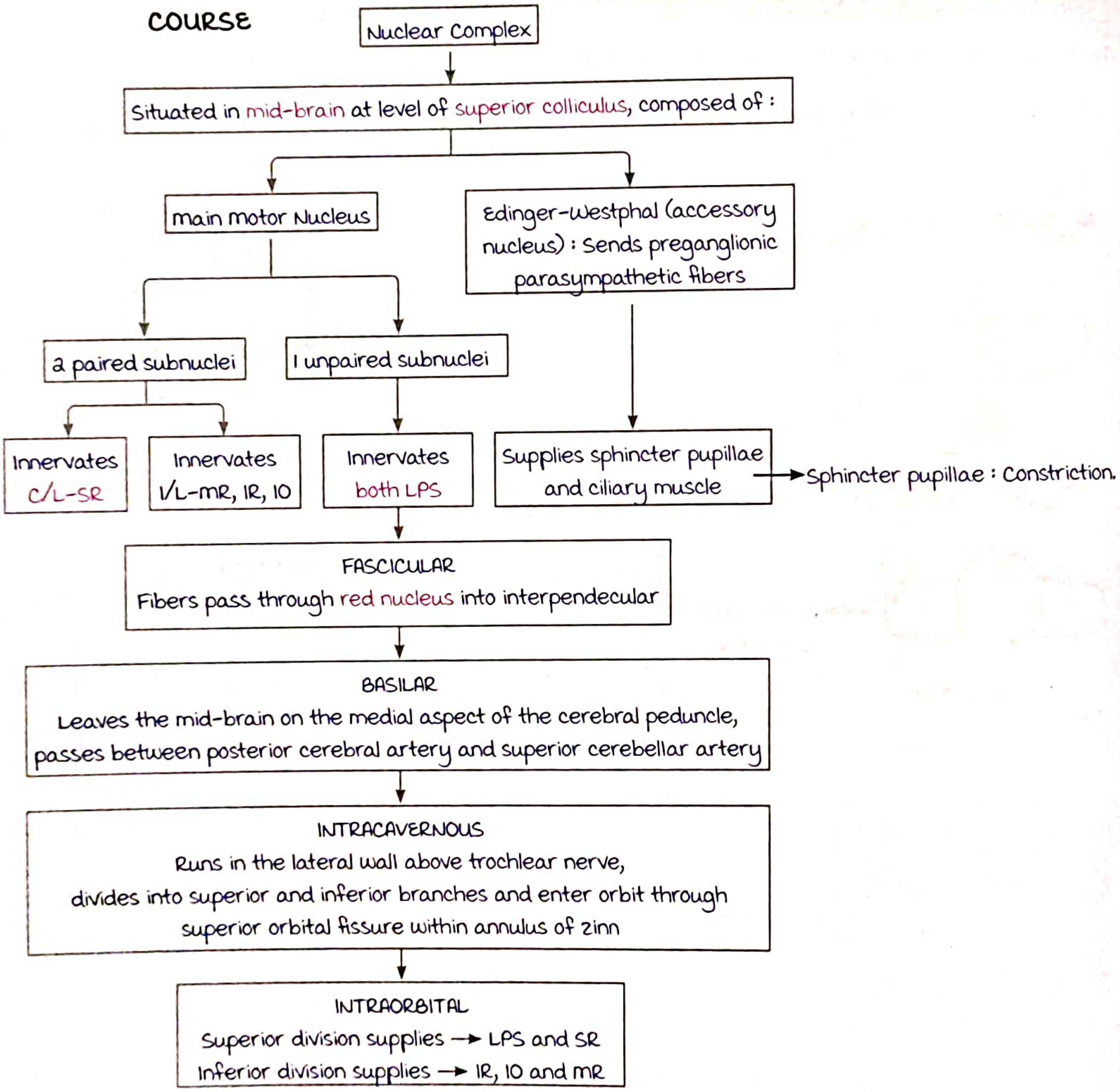
- Vertigo
- Deviation : Opposite to the action of paralysed muscle.
- 2° deviation > 1° deviation

Cause : 3rd, 4th, 6th nerve palsy.

III Cranial Nerve Palsy/Oculomotor Nerve Palsy

00:04:58

COURSE



Nuclear Complex

Situated in mid-brain at level of superior colliculus, composed of :

Main motor Nucleus

Edinger-Westphal (accessory nucleus) : Sends preganglionic parasympathetic fibers

2 paired subnuclei

1 unpaired subnuclei

Innervates C/L-SR

Innervates I/L-MR, IR, IO

Innervates both LPS

Supplies sphincter pupillae and ciliary muscle → Sphincter pupillae : Constriction.

FASCICULAR
Fibers pass through red nucleus into interpendecular

BASILAR
Leaves the mid-brain on the medial aspect of the cerebral peduncle, passes between posterior cerebral artery and superior cerebellar artery

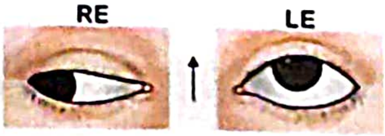
INTRACAVERNOUS
Runs in the lateral wall above trochlear nerve, divides into superior and inferior branches and enter orbit through superior orbital fissure within annulus of zinn

INTRAORBITAL
Superior division supplies → LPS and SR
Inferior division supplies → IR, IO and MR

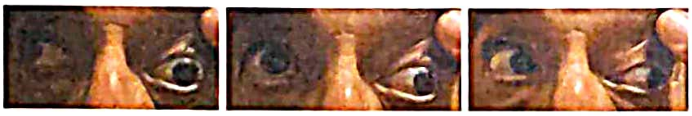
CLINICAL FEATURES

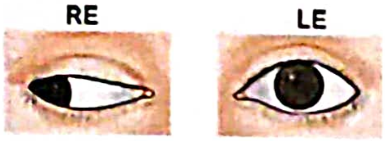
Muscle Involved	Normal Action	Clinical manifestation
Levator palpebrae superioris	Elevation of upper eyelid	Drooping of eyelid : Ptosis
Superior oblique / Inferior rectus	Elevation of eye	Downward deviation : Hypotropia
medial rectus	Adduction	Outward deviation : Exotropia ↓ Crossed diplopia
Sphincter pupillae	miosis	mydriasis with loss of pupillary light reflex
Ciliary muscle	Accommodation	Loss of near vision / Near reflex

O/E

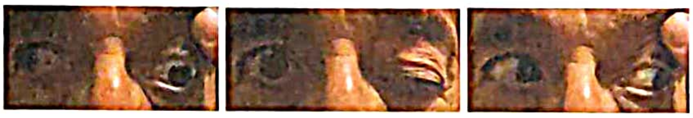


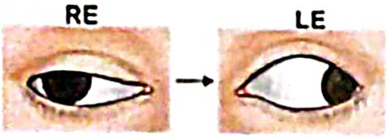
→ Limited elevation of LE ←



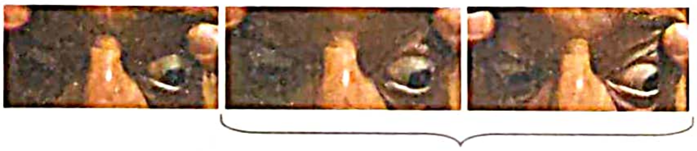


→ Primary gaze
- LE ptosis +
- LE down & out ←

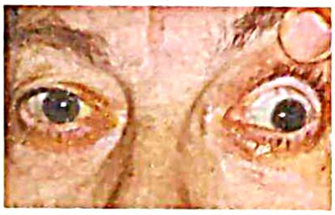




→ Limitation of Adduction ←



Down & outward movement present
↓

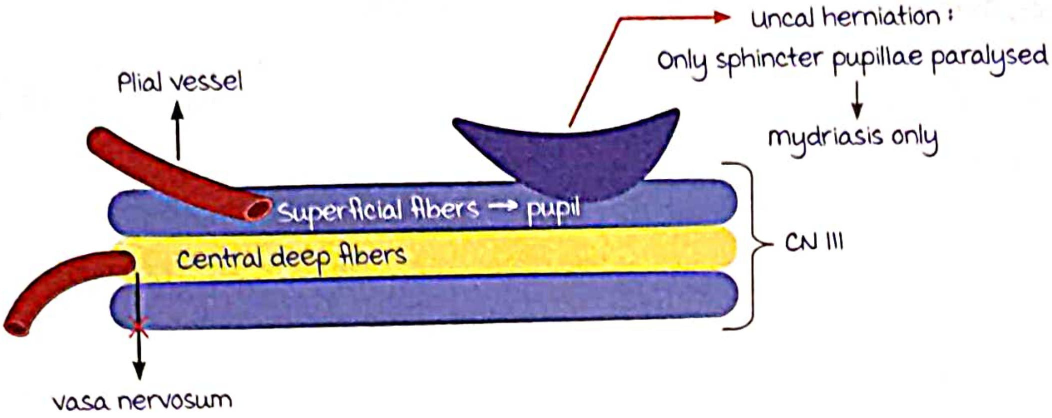


Special clinical features :

Seen in

- Vascular pathologies
- Uncal herniation
- Benedikt's syndrome :
  - lesion at red nucleus
  - CNIII palsy + flapping tremors
- Weber's syndrome :
  - lesion at cerebral peduncle
  - CNIII palsy + C/L hemiparesis
- Nothangel's syndrome :
  - CNIII palsy + cerebral ataxia
- Nuclear CN III palsy
  - ↓ Daroff's rule :
    - B/L ptosis
    - C/L SR weakness
    - B/L mydriasis & cycloplegia

----- Active space -----


Uncal herniation :
Only sphincter pupillae paralysed
mydriasis only
Plial vessel
Superficial fibers → pupil
Central deep fibers
CN III
vasa nervosum

Vascular pathologies :

- Caused by diabetes, hypertension
- Clinical features :
  - CN III palsy with pupillary sparing
  - Near reflex absent
  - Light reflex spared

TREATMENT

Observation : —— 6 months ——→ Surgery :
If no regeneration occurs.

- D/t regeneration by neurolemma
  - CNS : oligodendrocytes
  - PNS : Schwann cells

↓ If faulty

Abherrant regeneration :

- Clinical features :

Pseudo von Graffe sign
- Fibre of IR starts to supply LRS
↓
On downgaze, both IR & LPS contract
- Depression of eye (d/t IR)
- Elevation of upper lid (d/t LPS)

Lid Lag : Eyelid movement lags behind movement of eyeball

Pseudo Argyll Robertson Pupil

- Fibres of ciliary muscle supply Sphincter pupillae (S.P)
  ↓
  On near vision, Ciliary muscle : Accommodates S.P – miosis
  ↓
  Accommodation Reflex present
- Fibres of sphincter pupillae completely damaged
  ↓
  Pupillary light reflex absent

IV Cranial Nerve Palsy/ Trochlear Nerve Palsy

00:41:45

----- Active space -----

- A/K/A Superior Oblique Palsy.
- m/c congenital paralytic squint.

Course of CN IV :

Nucleus
midbrain at the level of inferior colliculus

↓

Fascicular
Curves around
aqueduct of sylvius and decussates

↓

Trunk
Leaves the midbrain on the dorsal surface,
passes between posterior cerebral and superior cerebellar arteries

↓

Cavernous
Runs in the lateral wall between oculomotor nerve and ophthalmic (branch of trigeminal)
nerve, passes into orbit through superior orbital fissure outside the annulus of Zinn

↓

Intraorbital
Innervates contralateral superior oblique

Clinical features :

Features are opposite to the action of the muscle paralyzed.

Normal action of SO	Manifestation upon palsy	Head posture of patient in palsy
Intorsion	Outward rotation : Excyclotropia	Head tilt to opposite side.
Depression	I/L Hypertropia	Chin down
Depression in Adduction	↑ Hypertropia on adduction, vertical diplopia on downgaze.	Face turns to opposite side

Note : C/L Hypertropia occurs only if lesion is at nucleus.

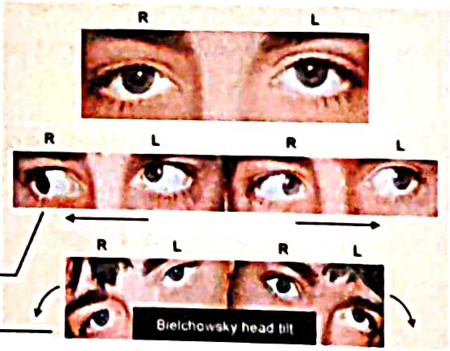
Diagnosis :

Park's 3-step test for SO palsy :

Step 1 : Identification of Hypertropia (LE).

Step 2 : Hypertropia worse on opposite gaze.

Step 3 : Hypertropia better on opposite tilt.



LE Hypertropia ↑ on right gaze

LE Hypertropia ↓ on RT

Parks 3 step test

----- Active space -----

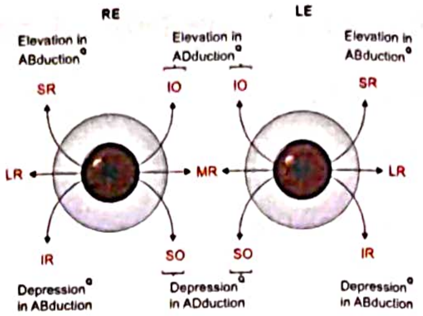
Note : Cyclovertical muscle Palsy (SR, IR, SO, IO)

To diagnose, follow Park's 3 step test

1. Identify muscles causing hypertropia.
2. Identify muscles causing lateral gaze.
3. Identify muscles causing head tilt.

$\underbrace{\hspace{6cm}}$

muscle implicated in all of the above is the affected muscle.


RE
LE
Elevation in ABduction^{Q}
Elevation in ADduction^{Q}
Elevation in ABduction^{Q}
SR
IO
IO
SR
LR
MR
LR
IR
SO
SO
IR
Depression^{Q} in ABduction
Depression^{Q} in ADduction
Depression^{Q} in ABduction

VI Cranial Nerve Palsy/Abducens Palsy/Lateral Rectus Palsy 01:10:58

Supplies lateral rectus.

Course :

Nucleus
Situated in pons^{Q}, ventral to floor of 4th ventricle

↓

Fascicular
Leaves brainstem at pontomedullary junction

↓

Basilar
Enters basilar cistern and crossed by anterior inferior cerebellar artery, passes through Dorello canal^{Q} and enters cavernous sinus

↓

Intracavernous
Runs inferomedially in the lateral wall, in close relation to ICA^{Q}
↓
Enter orbit through superior orbital fissure within annulus of Zinn

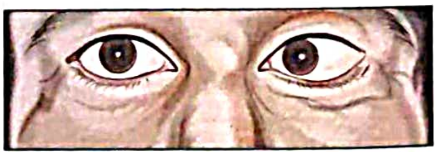
↓

Intraorbital
Innervates Ipsilateral lateral rectus

Note :

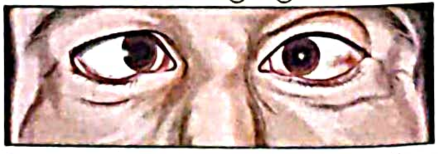
Nuclei of CN	Location
III, IV	midbrain
V, VI, VII, VIII	Pons
IX, X, XI, XII	medulla

RE LE
Abducens nerve (CN VI) palsy



1° gaze: LE esotropia

Normal Right gaze



Left gaze : Left abduction absent/limited

Causes :

Syndromes affecting pons :

Foville syndrome : IL 5/6/7/8 CN palsy + central Horners syndrome.

Millard-Gubler syndrome : IL 6/7 CN palsy + C/L hemiplegia.

Mobius syndrome : B/L 6/7 CN palsy.

Clinical features :

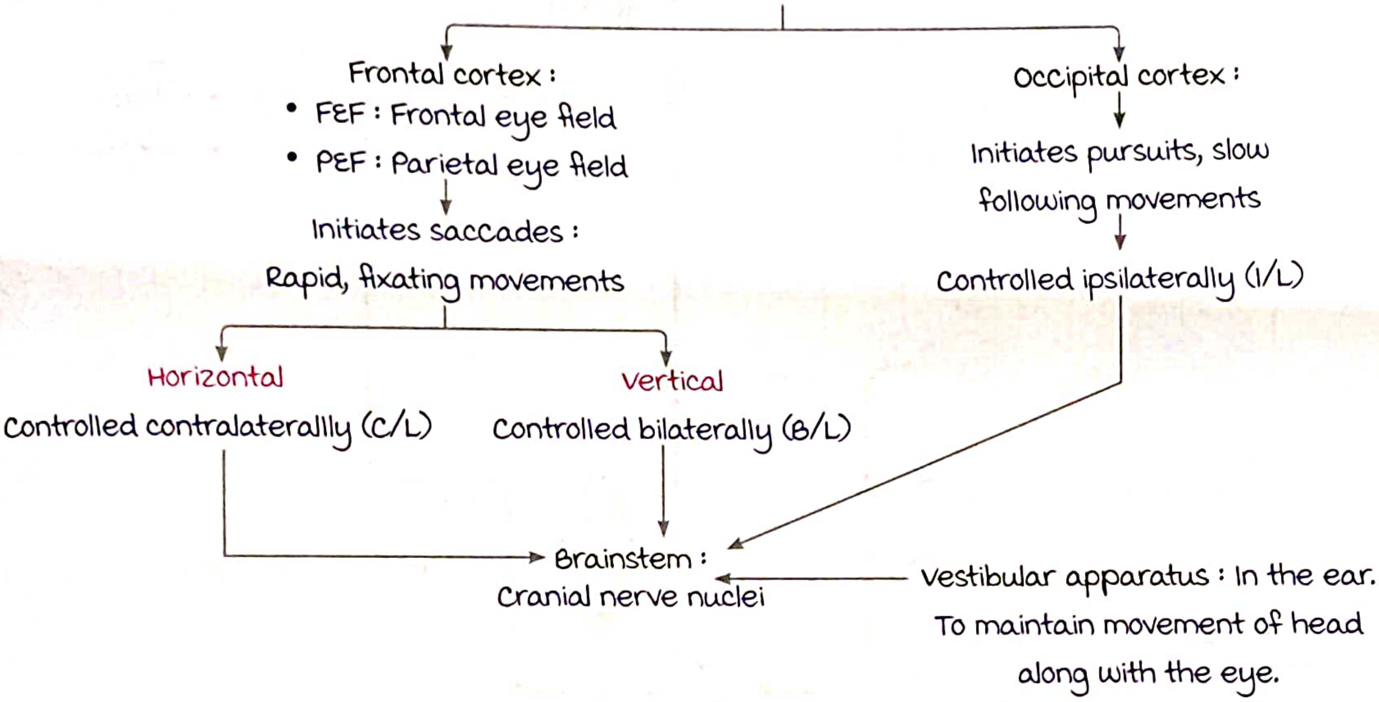
1. Limitation of Abduction → Inward deviation (LE esotropia).
2. Diplopia → Uncrossed type.
3. I/L Head posture.
4. I/L Horizontal gaze palsy.

GAZE DEFECTS

Supranuclear Control of Ocular Movements

00:00:30

Cerebral cortex :



Frontal cortex :
- FEF : Frontal eye field
- PEF : Parietal eye field

↓ Initiates saccades : Rapid, fixating movements

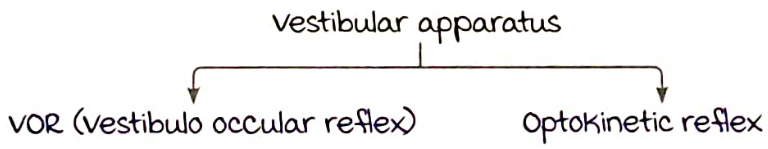
Horizontal: Controlled contralaterally (C/L)

Vertical: Controlled bilaterally (B/L)

Occipital cortex : ↓ Initiates pursuits, slow following movements ↓ Controlled ipsilaterally (I/L)

Brainstem : Cranial nerve nuclei

Vestibular apparatus : In the ear. To maintain movement of head along with the eye.



Vestibular apparatus
- VOR (Vestibulo occular reflex)
- Optokinetic reflex

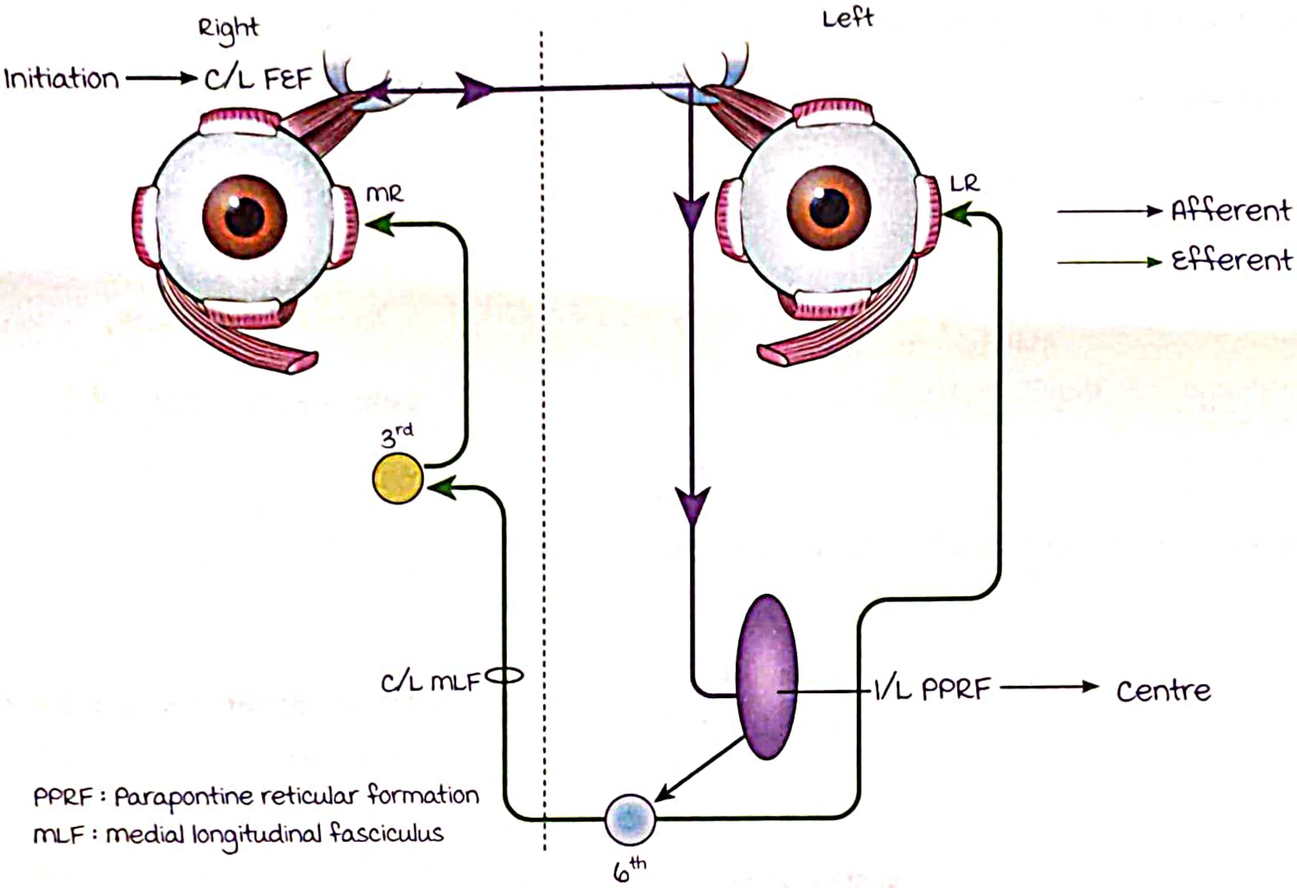
- Tests :
  - a. Oculo-cephalic reflex : AKA Doll's reflex.
    - Test CN III, VI, VIII.
    - Eyes move opposite to the movement of head (In coma patients).
  - b. Calorie test : Injection of water into ear.
    - Cold water : Opposite side nystagmus.
    - Warm water : Same side nystagmus.

----- Active space -----

Horizontal Gaze

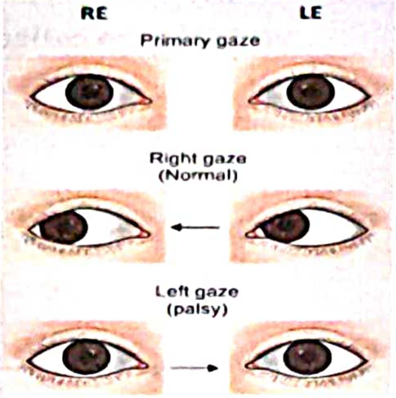
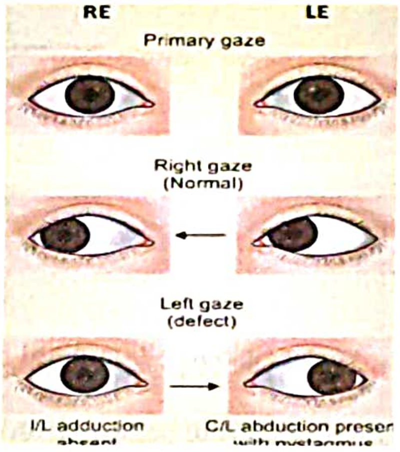
00:08:07

- Centre : Ipsilateral PPRF
- Saccadic movements to look towards left/right side


Right
Left
Initiation
C/L FEF
MR
LR
Afferent
Efferent
3rd
C/L MLF
I/L PPRF
Centre
6th
PPRF : Parapontine reticular formation
MLF : medial longitudinal fasciculus

Pathway of left gaze

Horizontal gaze palsy :

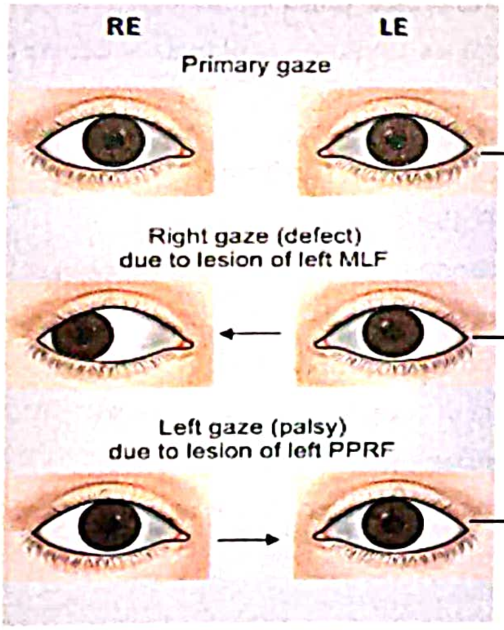
Lesion at	I/L LR	C/L MR	Features	Appearance
C/L FEF	Fails to contract	Fails to contract	Left gaze palsy	 RE LE Primary gaze Right gaze (Normal) Left gaze (palsy)
I/L PPRF	Fails to contract	Fails to contract	Left gaze palsy	
C/L MLF : Internuclear ophthalmoplegia ↓ m/c cause : multiple sclerosis	Can contract	Fails to contract	Left gaze defect : • I/L adduction absent • C/L abduction present	 RE LE Primary gaze Right gaze (Normal) Left gaze (defect) I/L adduction absent C/L abduction preser with nystagmus

One & a half syndrome :

I/L MLF → C/L gaze defect

I/L PPRF → I/L gaze palsy

→ 1 1/2 defect


RE
LE
Primary gaze
Right gaze (defect)
due to lesion of left MLF
Left gaze (palsy)
due to lesion of left PPRF

Normal

Partial loss of left gaze

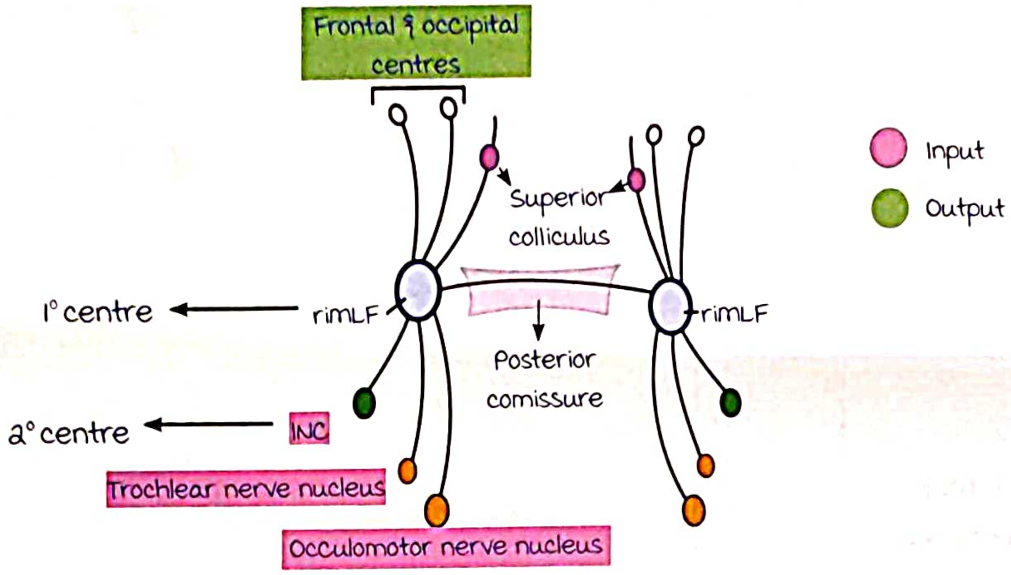
Completely loss of left gaze

Vertical Gaze

00:25:05

Eye movement in upgaze/downgaze.

Vertical gaze pathway :


Frontal & occipital centres
Superior colliculus
1° centre
rimLF
rimLF
Posterior comissure
2° centre
INC
Trochlear nerve nucleus
Occulomotor nerve nucleus
Input
Output

- rimLF : Rostral intestitial medial longitudinal fasiculus.
- INC : Intestitial nucleus of Cajal.

Etiology of vertical gaze palsy :

1. Distal basilar artery ischemia (m/c) :

   Supplies rimLF.

2. Progressive supranuclear palsy :
   - Palsy : Downgaze → upgaze → Global → Convergence.
   - Occurs in old age.

3. Parinaud's dorsal midbrain syndrome :
   - Site of lesion : Posterior commissure.
   - most common cause : Pinealoma > hydrocephalus.
   - Clinical features :
     - Upgaze palsy (supranuclear).
     - Collier's sign : upper eyelid retraction.
     - Convergence : Retraction nystagmus.
     - Light near dissociation : Light reflex absent & near reflex present.

RESTRICTIVE SQUINT AND COMITANT SQUINT

Restrictive Squint

00:00:10

Restrictive squint is d/t

Congenital causes :

- DRS : Duane retraction syndrome.
- Brown's syndrome.
- CFEOM (Congenital fibrosis of the extraocular muscles).

Acquired causes :

- Orbital trauma.
- Orbital tumours.

DUANE RETRACTION SYNDROME

Clinical features :

Type of DRS	a. Limitation of movement	b. Closure of palpebral fissure + c. Retraction of globe upon
1	Abduction	Adduction
2	Adduction	Adduction
3	Abduction & adduction	Abduction & adduction (D/t paradoxical innervation of both LR & MR)

Pathology :

Type I DRS :

- LR gets supplied by 3rd nerve instead of 6th nerve (Paradoxical)

On impulse for abduction via 6th nerve

↓

Lateral rectus does not act : No abduction

On impulse for adduction via 3rd nerve

↓

Both lateral rectus & medial rectus contract

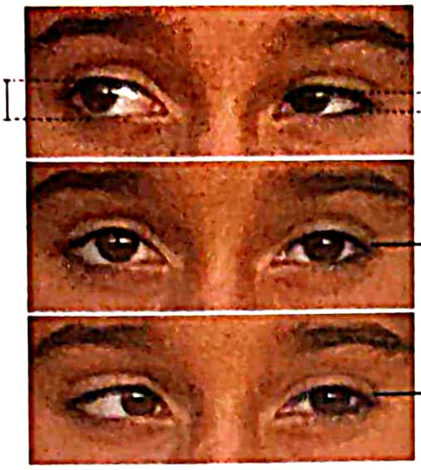
↓

Retraction of globe

↓

Closure of palpebral fissure

----- Active space -----



Right gaze : Left eye adduction present but with closure of left palpabral fissure

Primary gaze : Normal , No deviation

Left gaze : Left eye abduction limitation

Type I DRS of left eye

Diagnosis :

Force duction test.

BROWN'S SYNDROME

Congenitally tight superior oblique (SO) + ↓ movement of SO through trochlea (Bony hook)

↓

Contracture of superior oblique

↓

Superior oblique fails to relax

↓

Antagonist : Inferior oblique (IO) cannot relax

↓

Restriction of elevation in adduction.

Comitant Squint

00:17:47

m/c presentation : Esotropia (Convergent squint).

TYPES

- Accommodative
  - Refractive : Esotropia associated with hypermetropia.
  - Non refractive :
    - Due to high AC/A ratio.
      - D/t ↑ convergence or ↓accommodation.
      - (AC : Accommodative convergence, A : Accommodation)
      - 1 D of accommodation ~ 2° convergence.
    - Treatment :
      - miotics (Ecothiophate).
      - Bifocal spectacles.
      - Surgery :
        - a. Bimedial recession (Weakening of B/L medial recti).
        - b. Faden surgery.
- Non accommodative

Non accommodative esotropia :

AKA Essential infantile esotropia

Presentation : 4-6 months of age.

- No refractory error.
- No neurological deficit.

Associated with :

- Latent horizontal nystagmus.
- Inferior oblique overaction.
- Dissociated vertical deviation.
- Large angle deviation (>15°/>30 pd) → Confusion → Amblyopia of deviated eye.

Prevention of amblyopia :

Occlusion therapy : Intermittent alternate patching of normal eye.

- Patch normal eye for x days ⇄ Patch deviated for days (x : Age in years).
- Penalization : Use of Atropine drops in normal eye.

Note : Prevention of amblyopia can be done till 6 years of age (As eyeball completes its development by 6-7 years of age).

Surgery (TOC) : usually done at 1 year of age.

In the case of esotropia : $\underbrace{\text{Recession of I/L MR}}_{\substack{\text{Lengthening} \\ \downarrow \\ \text{Weakening.}}} + \underbrace{\text{Resection of I/L LR}}_{\substack{\text{Shortening} \\ \downarrow \\ \text{Strengthening.}}}$

PSEUDO-STRABISMUS AND OCULAR MYOPATHIES

Pseudo Strabismus

00:00:15

Apparent deviation.

2 types :

Pseudo eso

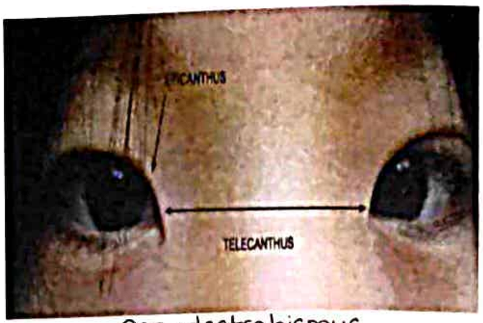
Causes :

1. Telecanthus
   ↑ ICD + normal IPD.
2. Epicanthus :
   An extra (Congenital) fold of skin connecting upper eyelid to lower eyelid in the medial part.
3. myopia.

Pseudo exo

Causes :

1. Hypertelorism
   - Increased ICD.
   - Increased IPD (D/t wide set orbits).
2. ROP (Retinopathy of prematurity)
   D/t temporal displacement of macula.
3. Hypermetropia.



Pseudostrabismus

Ocular Myopathies

00:09:55

MYASTHENIA GRAVIS

m/c in young females.

Pathology :

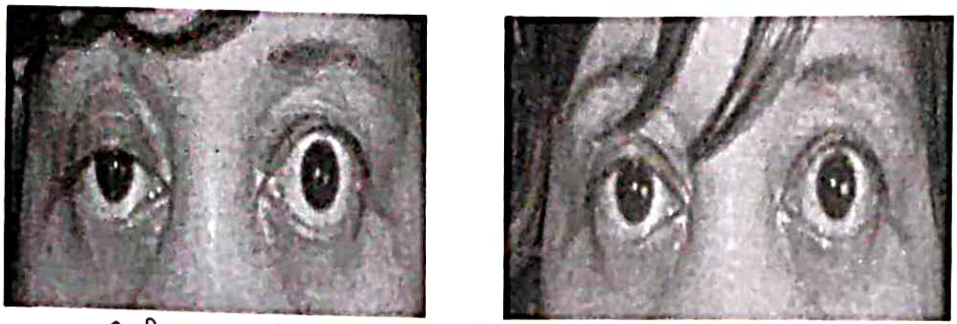
- Reduced Ach receptors.
- Fatigue : worsens in the evening (m/c feature).

Ocular features :

a. Ptosis : B/L.
b. Diplopia.
c. Cogan lid twitch sign : upshoot of eyelid when changing gaze from down to up.
d. Orbicularis oculi weakness : Tested by forced opening of closed eyelids.

Diagnosis :

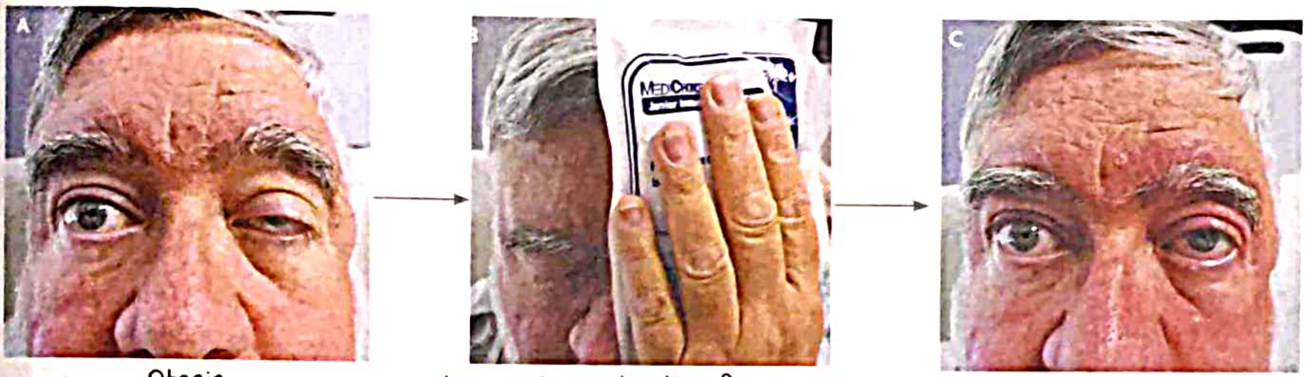
1. Tensilon test : Inj 0.2 mg of edrophonium (Anticholinesterase agent) → ↑ Ach.



Before injection

After injection → Improvement of ptosis

2. Ice-pack test :



Ptosis

Ice pack application for 5 mins

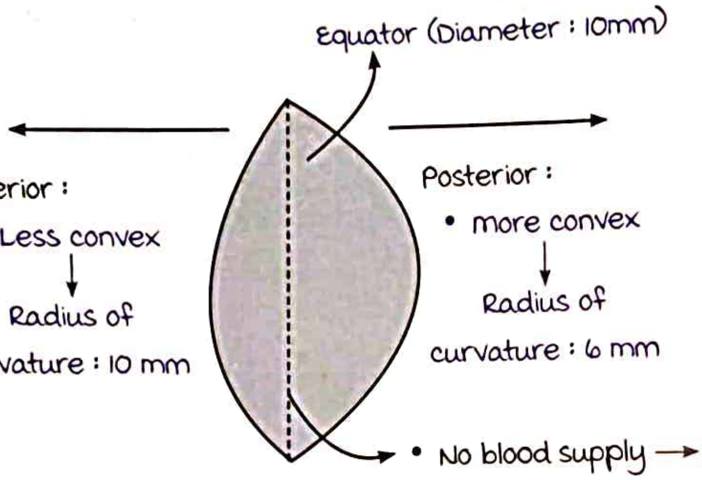
Ptosis improvement

ANATOMY AND METABOLISM

00:01:13

Functional Anatomy of Lens

Structure of the lens :



Anterior :

- Less convex ↓ Radius of curvature : 10 mm

Posterior :

- more convex ↓ Radius of curvature : 6 mm

Biconvex lens

Site : Patellar fossa (Between anterior & posterior segment).

- No blood supply → Transparency.
- No nerve supply.

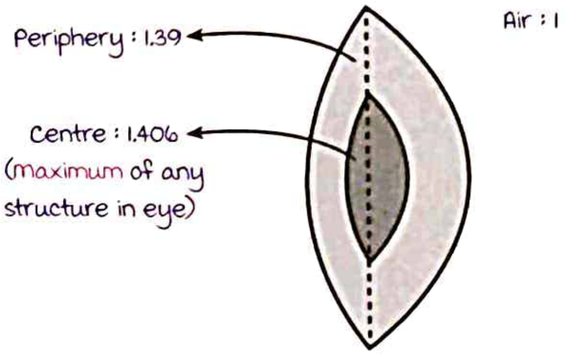
(Change in curvature during accommodation is d/t zonules)

Function of lens :

Converging refraction of light.

Refractory power :

- 16 D (Far) +19 D (Near).
- Variable d/t accommodation.



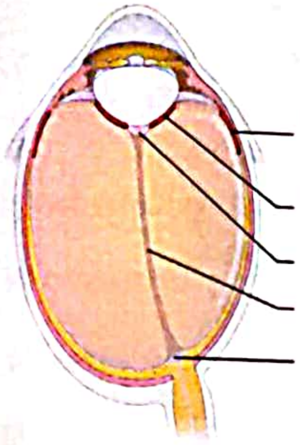
Periphery : 1.39

Centre : 1.406 (maximum of any structure in eye)

Refractory indices

Attachments of the lens :

On the posterior surface :


Vitreous base
Weigert's ligament
Berger's space
Cloquet's canal
Space of Martegian

Weigert's ligament → Circular attachments of lens § anterior hyaloid/vitreous.

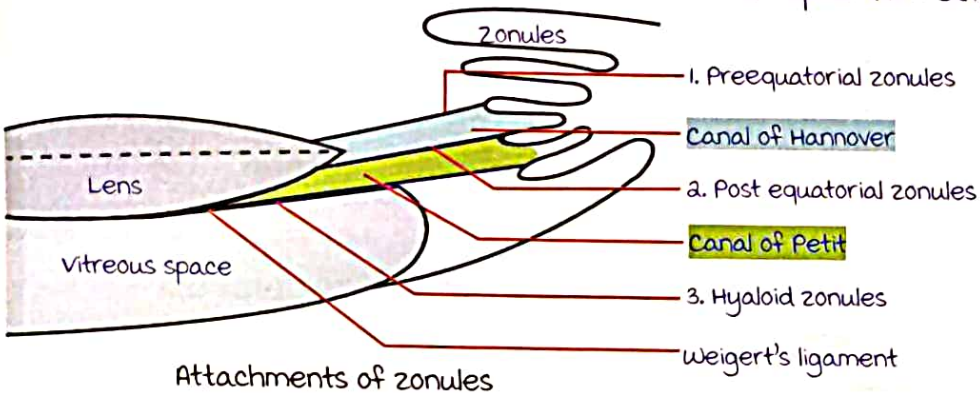
Berger's space → Retrolental cavity central to Weigert's ligament.

Cloquet's canal → Potential space during embryogenesis : Contained hyaloid artery to supply developing lens § vitreous.

↓

Disappears by 8 months of gestation.

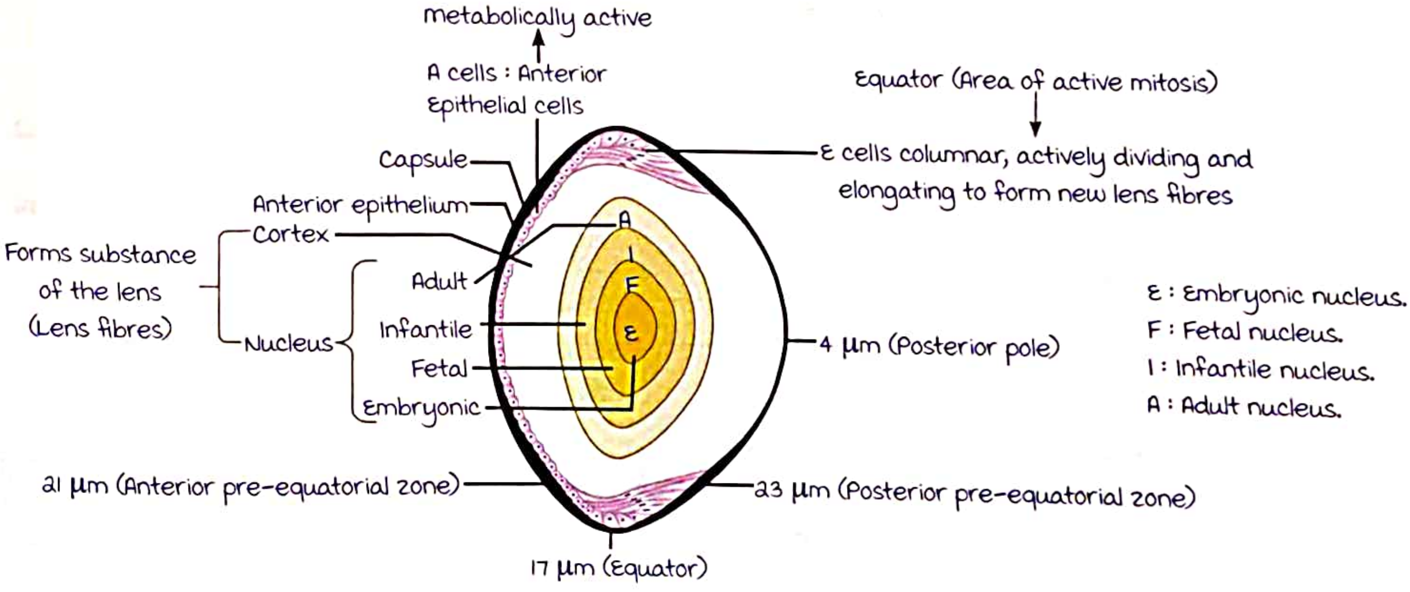
- If remnants are seen :
  - -On posterior lens : mittendorf dots.
  - -On optic disc : Bergmeister papillae.


Zonules
Lens
Vitreous space
1. Preequatorial zonules
Canal of Hannover
2. Post equatorial zonules
Canal of Petit
3. Hyaloid zonules
Weigert's ligament

Attachments of zonules

Structure Of The Lens

00:17:50


metabolically active
A cells : Anterior Epithelial cells
Equator (Area of active mitosis)
ε cells columnar, actively dividing and elongating to form new lens fibres
Capsule
Anterior epithelium
Cortex
Forms substance of the lens (Lens fibres)
Nucleus
Adult
Infantile
Fetal
Embryonic
A
I
F
ε
4 µm (Posterior pole)
21 µm (Anterior pre-equatorial zone)
23 µm (Posterior pre-equatorial zone)
17 µm (Equator)

ε : Embryonic nucleus.
F : Fetal nucleus.
I : Infantile nucleus.
A : Adult nucleus.

Capsule :

- Outermost/protective layer.
- Thickest basement membrane in the body.
- Composed of type IV collagen.
- Variable thickness of capsule :
  - Thinnest : Posterior pole of the capsule.
  - Thickest : Posterior pre-equatorial zone.

Anterior epithelium :

- Contains 2 types of cells :
  - A cells.
  - E cells.

Posterior epithelium :

- Absent in adults.
- Formed primary fibres during embryogenesis.

Cortex :

- Formed by E cells & posterior epithelium.

Nucleus :

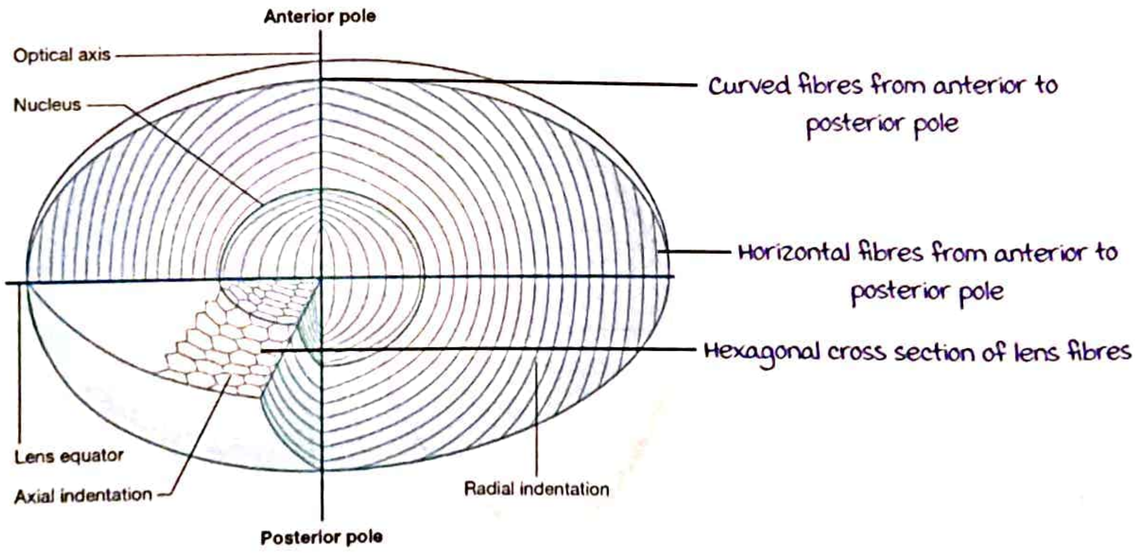
	Embryonic nucleus	Fetal nucleus	Infantile nucleus	Adult nucleus
Time of formation	Day 27 of fertilization (Oldest lens fibres)	During implantation on the uterine wall	Immediately after birth	1 month after birth (Youngest lens fibers)
Lens fibres	Primary	Secondary		

Sutures in lens/interdigitations :

- Seen in fetal nucleus.
- D/t its formation & interdigitations with the embryonic nucleus.

	Anterior	Posterior
Shape	Y	λ (Inverted Y)
Interdigitations	Apical processes	Basal processes

Lens matrix architecture :


Anterior pole
Optical axis
Nucleus
Curved fibres from anterior to posterior pole
Horizontal fibres from anterior to posterior pole
Hexagonal cross section of lens fibres
Lens equator
Axial indentation
Radial indentation
Posterior pole

Note :

- Sequestered antigen : Lens protein released from the capsule during surgery/ trauma to the lens.
- Ascorbic acid transport in lens epithelium : mediated by $SVCT_2$ (Sodium dependent vitamin C transporter).

Biochemistry Of Lens

00:35:08

PROTEINS IN THE LENS

Water-soluble proteins :

a. Crystallins :

α-crystallins :
- most important.
- Heat shock proteins prevents denaturation.

β-crystallins :
- maximum amount.

γ-crystallins.

b. High molecular weight proteins (HMWP) :
- HM1.
- HM2.

Water insoluble proteins :

a. Urea solubilitity :

Urea soluble :
- Vimentin/Filensin (Crystal proteins)

Urea insoluble :
- MIP 26 : AKA Aquaporin 0.
- Maintains transparency of lens by maintaining hydration.

b. HMWP : HM3, HM4 (↑ in cataract).

ANTIOXIDANT MECHANISMS IN LENS

Antioxidants :

- Vitamin C.
- Vitamin E.
- Glutathione reductase.
- Superoxide dismutase.
- Catalase.
- Vitreous : maintains low O_2 level around lens (Removal : ↑ propensity for cataract).

Note :

- Vitamin A does not act as antioxidant in lens.
- Tryptophan deficiency

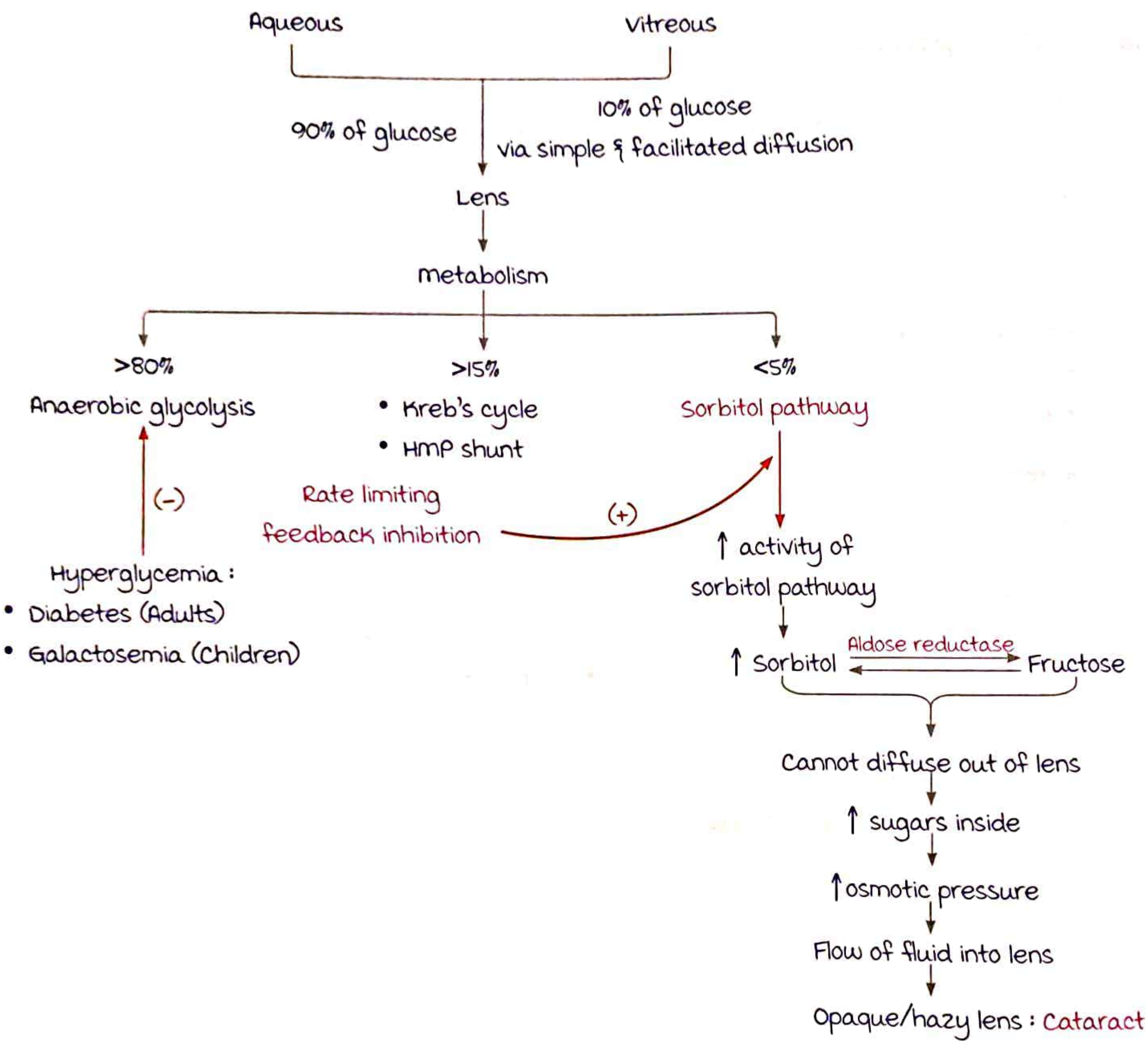
↓

↓ level of Kynurenine and antioxidants (Except SOD)

↓

↑ incidence of cataract (Based on experimental model in animals)

METABOLISM IN LENS



Aqueous → 90% of glucose → Lens

Vitreous → 10% of glucose via simple & facilitated diffusion → Lens

Lens → metabolism

metabolism:

- >80% Anaerobic glycolysis
- >15%
  - Kreb's cycle
  - HMP shunt
- <5% Sorbitol pathway

Hyperglycemia :

- Diabetes (Adults)
- Galactosemia (Children)

Hyperglycemia → (–) Anaerobic glycolysis

Rate limiting feedback inhibition → (+) Sorbitol pathway

Sorbitol pathway → ↑ activity of sorbitol pathway → ↑ Sorbitol $\underset{}{\overset{\text{Aldose reductase}}{\rightleftharpoons}}$ Fructose

→ Cannot diffuse out of lens → ↑ sugars inside → ↑ osmotic pressure → Flow of fluid into lens → Opaque/hazy lens : Cataract

ACQUIRED CATARACT : TYPES

Overview

00:00:20

Any opacity in the lens or its capsule.

Clinical features :

Gradual, painless loss of vision →
Blindness (m/c cause in India).

Note :

Loss of vision :

- Gradual, sudden.
- Painless, pain (Signs of inflammation).

Classification :

Acquired :

- metabolic.
- Traumatic.
- Complicated.
- Senile/age related.

Congenital :

- Infantile (Onset <1 year).
- Developmental (Onset >1 year).

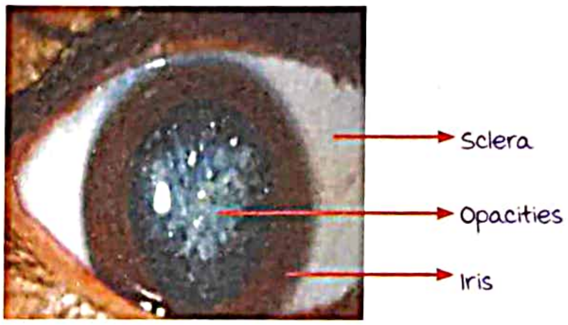
Metabolic Cataract

00:04:45

DIABETES MELLITUS

A. True diabetic cataract/Snow flake cataract :

- Cortical cataract.
- Type I DM.



Snowflake cataract

B. Pre-senile cataract :

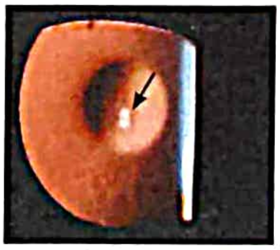
- m/c in type 2 DM.
- Senile cataract before 50 yrs of age.
- D/t carbamylation of crystallins.

Note : Other conditions of eye d/t DM.

- Fluctuating refractory errors :
  - D/t fluctuations in the glycemic state.
- Diabetic retinopathy :
  - Nonproliferative and proliferative

GALACTOSEMIA

- Oil droplet cataract/posterior subcapsular cataract.
- Only reversible cataract.
- Seen in infancy.



Oil drop cataract

Etiology :

Galactose ——×——→ Glucose.

D/t autosomal recessive metabolic conditions like deficiency of :

----- Active space -----

a. Galactose-1-PUT (Phosphate uridyl transferase)
b. UDP galactose epimerase.
} Classical galactosemia.

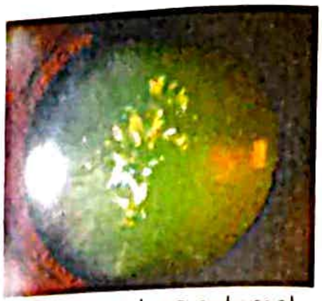
c. Galactokinase :
  - Galactose (Substrate) & galactitol/dulcitol (metabolites) accumulate in lens resulting in cataract.

Diagnosis :

Urine assay : measurement of increased reducing substances after breast feeding.

MYOTONIC DYSTROPHY

- Christmas tree cataract/posterior subcapsular cataract.
- Associated ocular manifestation :
  - Ptosis (Drooping of upper eyelid).
  - Diplopia.



Christmas tree cataract (Scattered, fine, iridescent irregular opacities)

WILSON'S DISEASE

Lens :

- Sunflower cataract.
- D/t deposition of cuprous oxide which can occur at multiple sites.

Cornea Kayser-Fleischer ring :

A golden brown ring d/t deposition of copper to descemet's membrane (Not pathognomonic of wilson's).

Seen in :

a. All case with neurological involvement (Basal ganglia Cu deposition).
b. Only 50% cases with hepatic involvement (Hepatolenticular degeneration).
c. Primary biliary cholangitis.
d. Neonatal hepatitis.

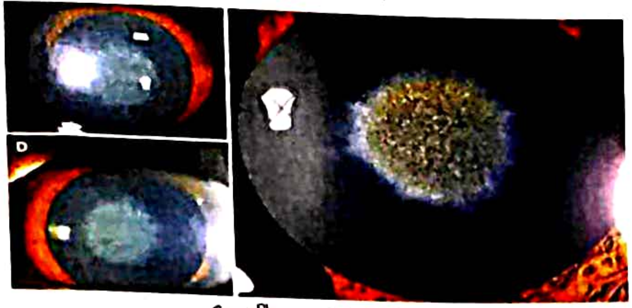
Progression :

- Appears at the superior cornea earliest → Inferiorly → Circumferential.
- missed clinically d/t being obscured by eyelid.

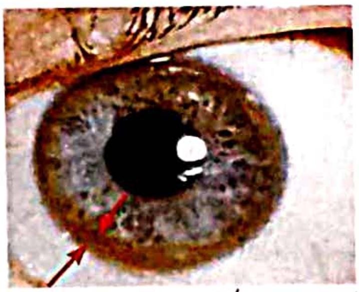
Treatment :

- Chelating agents :
  - Penicillamine and Trientine (Lifelong therapy).
  - MOA : Chelation of Cu → Cu load ↓ → ↓ Cu absorption.
  - KF ring usually disappears after 3-5 years.

- maintenance therapy :
  - Zinc acetate (Zinc copper complex ⟶ ↓ Cu absorption).



Sunflower cataract



Kayser-Fleischer ring : D/t copper deposists

PARATHYROID TETANY

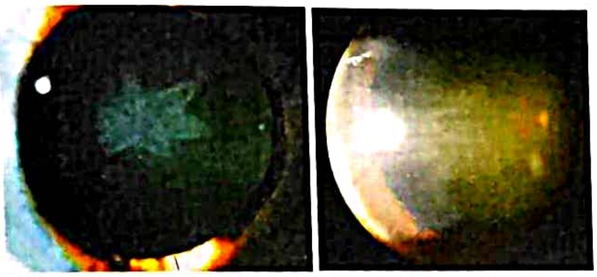
- Leads to hypocalcemia ⟶ Cataract.

	Children	Adults
Origin	Congenital	Acquired
Cataract location	Lamellar	Subcapsular

Traumatic Cataract

00:30:35

Blunt trauma :



Rosette shaped cataract : Posterior subcapsular

Lightning/electric shock :

Anterior capsular opacities.

Infrared rays :

- Highest wavelength rays.
- Glassblower's cataract (True exfoliation of lens capsule).

Penetrating trauma :

- Iron deposition in eye : Siderosis bulbi (D/t trauma with iron objects).
- Cortical cataract.

Complicated Cataract

00:37:40

Etiology :

Posterior subcapsular cataract caused secondary to another condition.

mnemonic : UMAR

a. Uveitis (m/c).

b. High myopia.

c. Angle closure glaucoma.

d. Retinitis pigmentosa : Pigment deposition in retina along with night blindness.

Appearance :

Breadcrumbs with polychromatic lustre.

ACQUIRED CATARACT : SENILE CATARACT

- Incidence : 50-70 years.
- Anticipation phenomenon present (Earlier occurrence if family history present).
- Increasing age → Oxidative damage to membranes & proteins.

Note :

Other risk factors for cataract formation :

- Sunlight (UV A + UV B) → Hydration.
- Use of corticosteroids.

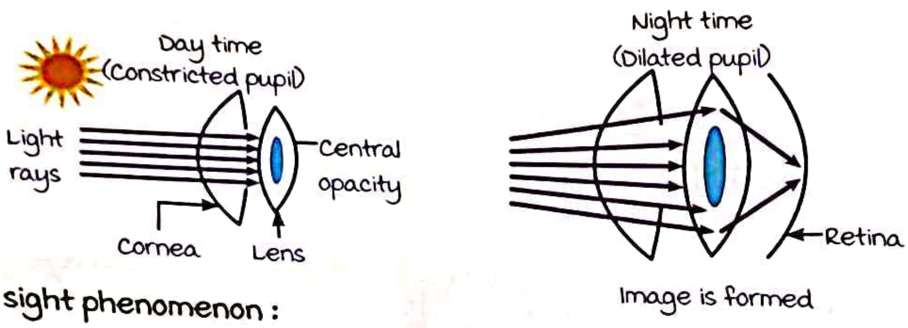
CLASSIFICATION

Based on site.

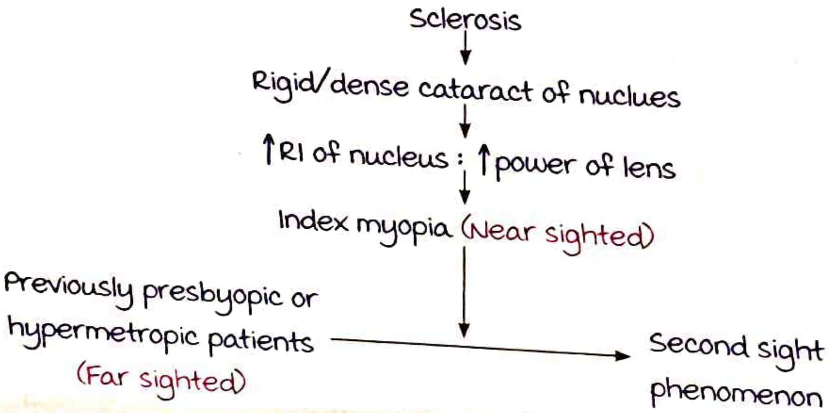
Nuclear Cataract

00:03:25

- AKA central cataract.
- Loss of vision : Daytime > night.


Day time
(Constricted pupil)
Light rays
Central opacity
Cornea
Lens
Night time
(Dilated pupil)
Retina
Image is formed

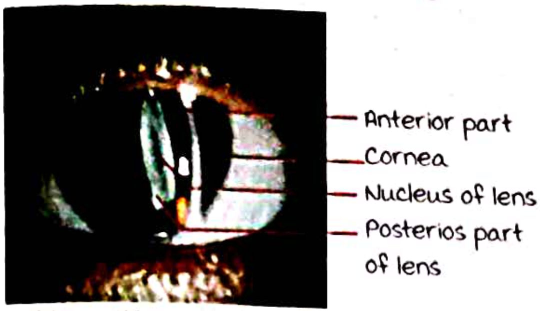
Second sight phenomenon :


Sclerosis
Rigid/dense cataract of nuclues
↑RI of nucleus : ↑power of lens
Index myopia (Near sighted)
Previously presbyopic or hypermetropic patients (Far sighted)
Second sight phenomenon

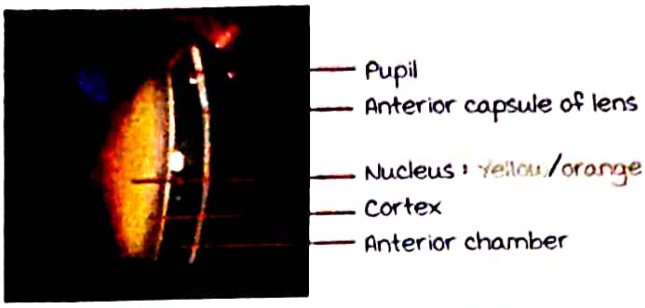
----- Active space -----

Grading :

- Grade 1 : Yellow (D/t urochrome pigment $\xrightarrow[\text{to}]{\text{Leads}}$ xanthopsia).
- Grade 2 : Amber.
- Grade 3 : Brown (Cataracta brunescens).
- Grade 4: Black (Cataracta nigra).


Anterior part
Cornea
Nucleus of lens
Posterios part of lens

Oblique slit illumination of normal lens


Pupil
Anterior capsule of lens
Nucleus : Yellow/orange
Cortex
Anterior chamber

Grade 2/3 cataract

Cortical Cataract

00:21:45

- Peripheral cataract.
- Loss of vision : Night time (Nyctalopia/night blindness) > daytime.

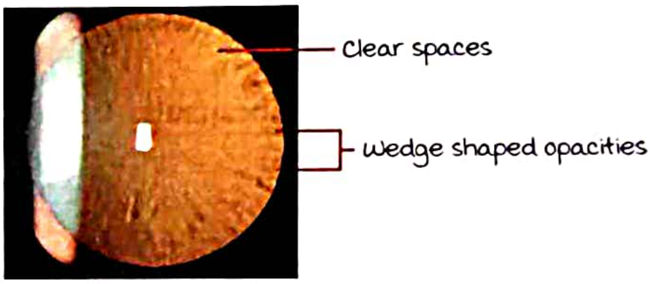
Progression :

Hydration : Water clefts
↓
Lamellar separation
↓
Cuneiform cataract
AKA immature senile cataract
- Greyish white colour lens.
- Presence of iris shadow.

↓ Continuous hydration/ swelling if not treated

Mature senile cataract/Intumescent cataract
- Pearly white colour, absence of iris shadow (D/t swollen lens).
- m/c complication : Phacomorphic glaucoma.

↓ If not treated


Clear spaces
Wedge shaped opacities

Cuneiform cataract : m/c inferonasally

Hypermature senile cataract —m/c complication→ Subluxation of lens

Complete disintegration of cortical fibres

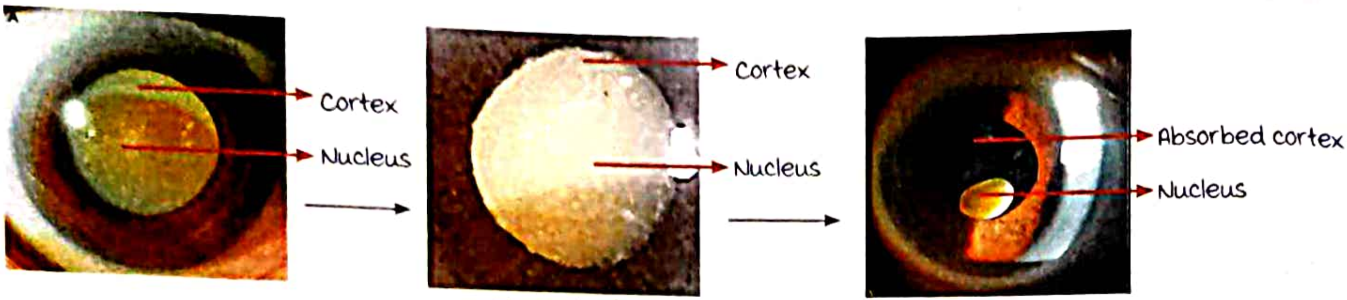
(D/t dysfunction of zonules)

↓ ↓

morgagnian cataract : > Sclerotic calcified cataract :

Liquefaction of cortex
↓
Sinking of nuclues
m/c complication :
Phacolytic glaucoma.

Sclerotic calcified cataract :
Disintegrated cortex
↓
Leak through capsule
↓
Shrinkage of lens with
thickened anterior capsule.



Variants of morgagnian cataract

Sclerotic calcified cataract :

Disintegrated cortex → Leak through capsule → Shrinkage of lens → With wrinkled, thickened anterior capsule.

Posterior Subcapsular Cataract (PSC) 00:44:25

- AKA cupuliform cataract.
- Posterior subcapsular cataract.

Clinical features :

a. Glare : C/o difficulty in driving at night d/t bright light.

b. Loss of vision :
  - Day time > night time.
  - Near vision > far.

c. maximum loss of vision of all the cataracts (Opacity at nodal point of eye).

Pathology :

Dysplasia of 'E' cells (Equatorial cells)
↓
Posterior migration & enlargement
↓
Bladder/Wedl cells : Collect to form plaque (Opacity)

Etiology :

a. Steroids :

- Systemic steroids : Cataract.
- Topical steroids : Glaucoma.

b. X-rays :

- Occupational exposure to ionizing radiation.

c. Other conditions :

- Neurofibromatosis type 2.
- myotonic dystrophy.
- Galactosemia.
- Blunt trauma.
- Complicated cataract : Primarily d/t uveitis/post uveitis.

ANTERIOR SUBCAPSULAR CATARACT

Rarest form of cataract.

Etiology :

mnemonic : AACG.

- Acute angle closure glaucoma : Glaukomflecken.
- Atopic dermatitis : Shield cataract.
- Amiodarone.
- Chlorpromazine.
- Gold deposits.

CATARACT SURGERY

PRE-OPERATIVE STEPS / PREPARATIONS

1. Dilate the pupil : Tropicamide + Phenylephrine eyedrops
2. Local Anaesthesia :

a. Topical : Proparacaine drops + b. Intracameral lidocaine augmentation

c. Peribulbar block > d. Retrobulbar block.

- mydricaine used :
    - Bupivacaine (long acting) + Lignocaine + 1 : 2,00,000 Adrenaline + Hyaluronidase (for better penetration).

e. Sterilize with Pre-operative Antibiotics

- Universal prophylaxis : 5% povidone iodine / betadine solution (instilled in the conjunctival sac).

Cataract Surgeries

00:05:45

PHACOEMULSIFICATION

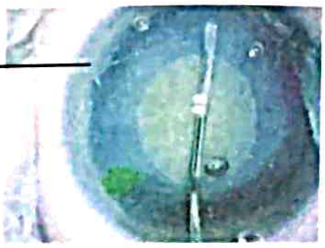
Part 1 :

1. Side Port incision : made with 15° blade / MVR Blade (2 cutting edges).
    - ≤ 1 mm in length.

1 cutting edge

15° blade

2. Staining of anterior capsule : Trypan blue

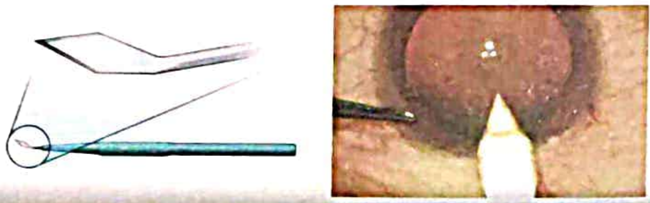


Trypan blue stained capsule

3. Continuous Curvilinear Capsulorhexis :
    - Hole made in the anterior capsule
    - Advantages over the Can opener technique (obsolete) :
        - Reduces radial tears.
        - Stabilizes the lens.
        - Decreases the chance of posterior capsule opacification (PCO).

Part 2 :

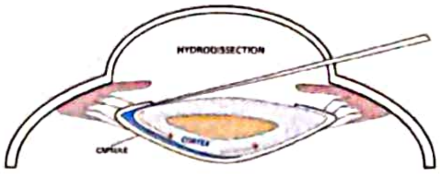
4. main post incision : using a 2.2mm Keratome
   (Size of Keratome : 2.2mm, 2.4mm, 3.2mm)



Keratome

----- Active space -----

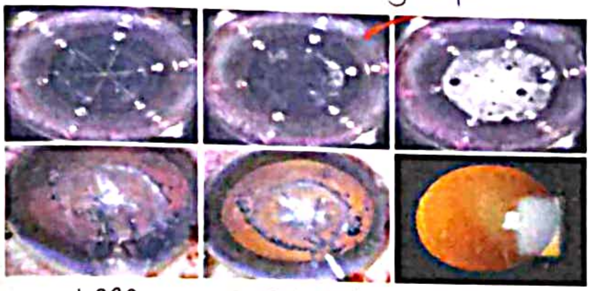
5. Hydrodissection : Separation of cortex from the capsule by injecting a jet of fluid below the margin of anterior capsule.
6. Hydrodelineation : Elective step.
   - Separation of epinucleus from endonucleus by a jet of fluid.
   - Advantage : Softening of epinucleus → easy aspiration along with cortex.



7. Nuclear fragmentation with phaco-probe :
   Piezoelectric tip of probe converts ultrasonic energy → to § fro mechanical motion.
8. Irrigation and aspiration of nuclear fragments § cortical matter.
9. Implantation of IOL
10. Incisions closed by hydration : no suture used.

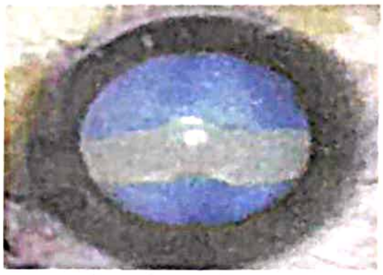
FLACS

- Procedure similar to that of Phacoemulsification.
- Laser used in the following steps :



1. CCC
2. main port incision
3. nuclear fragmentation

Note : Complication of Intumescent cataract:
↑ intralenticular pressure → radial tear during CCC.

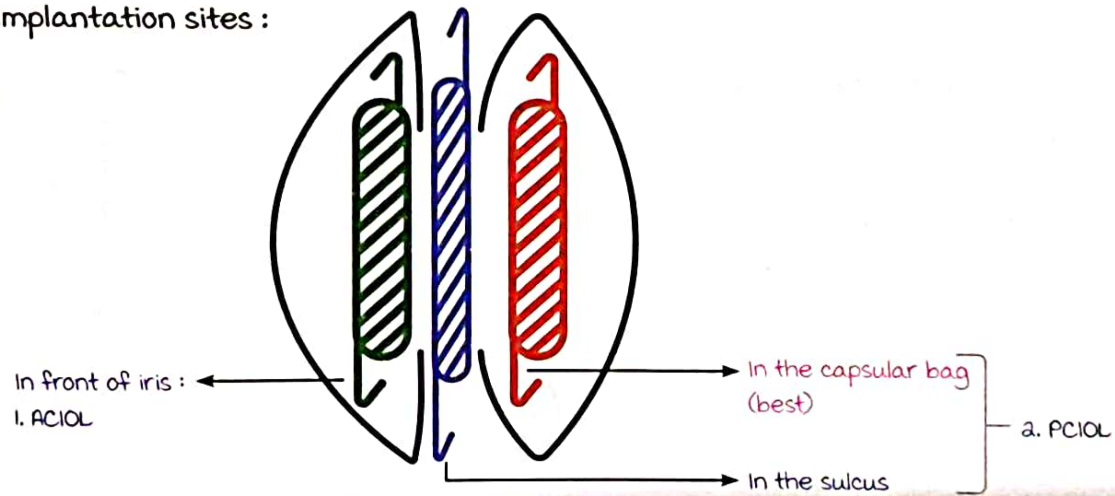


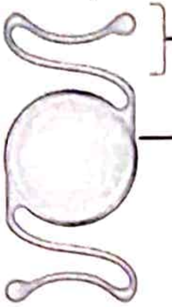
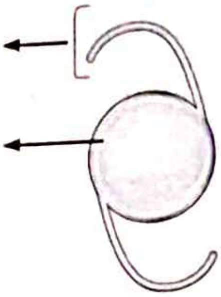
Argentinian flag sign

Intraocular Lens (IOL)

00:31:33

Implantation sites :

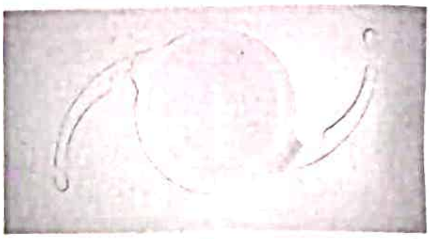


	ACIOL	PCIOL	
		Rigid type	Foldable
made of :	PMMA : Poly methyl methacrylate	PMMA	Acrylic : Hydrophilic Silicone : Preferred in vitreal surgeries
Shape of the IOL	S shaped 	C shaped 	

Note : Rigid lens → ICCE, ECCE ; Foldable lens → Phacoemulsification, FLACS.

Types of IOL :

1. multifocal IOL :
   - Has multiple rings for near, intermediate & far vision.
   - Side effect : leads to glare & halos in bright lights.
2. Toric IOL : In case of preexisting astigmatism/cylindrical power.
4. Square edged IOL : Decreases the chance of PCO.



multifocal IOL

Biometry (IOL) :

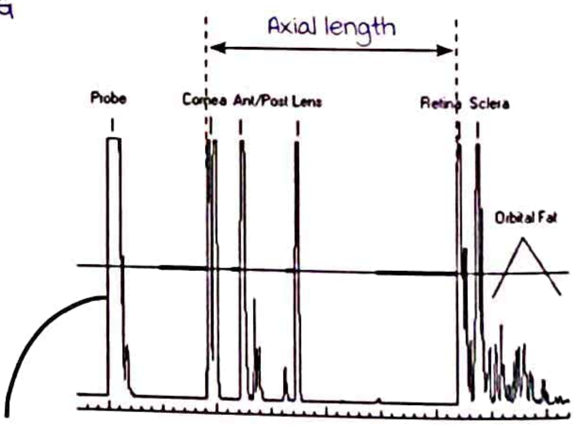
Calculation of IOL power for a patient pre-op.

Step 1 : Corneal curvature measurement by Keratometry.

Step 2 : Axial length of eyeball :
- Anteroposterior diameter : 24cm/24mm (Normal)
- measured by A [Amplitude] scan USG
  - Uses 8MHz sound waves

Step 3 : Formulae.

a) SRK – 7 Formula (m/c).
b) Holladay's Formula : If AL > 24.5 mm.
c) Hoffer Q Formula : If AL < 22 mm.
d) Haigis L Formula : Used in post LASIK cases
(D/t changes in corneal curvature).



A Scan

COMPLICATIONS OF CATARACT SURGERY

Complications

- Operative
- Acute post operative
- Late post operative

Operative Complications

00:00:10

POSTERIOR CAPSULAR RUPTURE (PCR)

Causes :

- D/t incomplete hydrodissection : Capsule aspirated along with cortex.
- Defective nuclear fragmentation.

Complications of PCR :

- Implantation disturbance.
- Fall of IOL into the vitreous.

} Resolved by implantation at :
  - Sulcus.
  - Anterior chamber.

- Vitreous in the anterior segment.

UGH SYNDROME

Uveitis - Glaucoma - Hyphema (Collection of blood in AC).

Causes :

ACOL implantation → Iris chafing.

Complications specific to ACIOL :

Iris chafing

- Inflammation : Uveitis
- Glaucoma : Pigment blocking the trabecular meshwork (Iris : Pigmented tissue)
- Hyphema : D/t vascularity of iris

Management :

- Steroids/antiglaucoma agents.
- Explant ACIOL (In severe cases).
- Slower prognosis : 7-10 days to regain vision (24 hours normally).

----- Active space -----

EXPULSIVE CHOROIDAL HAEMORRHAGE

Cause :

Pressure difference during surgery → Haemorrhage of choroidal vessels

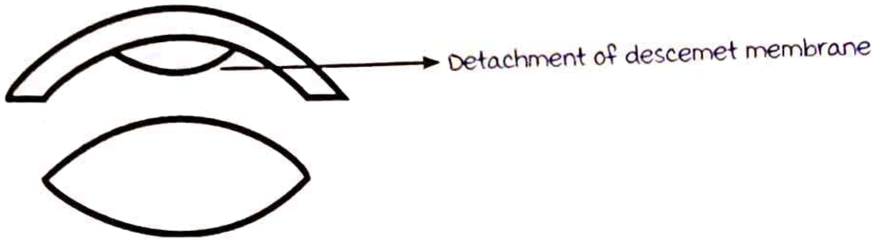
Short posterior ciliary artery

- Supplies :
  - Posterior portion of sclera, choroid
  - Outer4 layers of retina.

DESCEMET MEMBRANE DETACHMENT

Cause :

Trauma to the inner surface of the cornea d/t entry/exit of the instrument during surgeries.



Treatment : Descemetopexy → Air bubble in anteriorchamber.

Acute post operative complications

00:11:15

Iris prolapse :

D/t defective closure of incision in the periphery of cornea.

Endophthalmitis (EO) :

Suppurative inflammation of all layers of eyeball except sclera.

Etiology : Infective

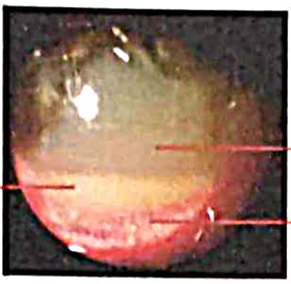
Agent	m/c cause of
Staphylococcus epidermidis/ Coagulase negative staphylococcus	Acute onset (<7 days) Post-op EO
Propionibacterium acnes	Late onset (6 weeks – 6 months) Post-op EO
Bacillus cereus	Post traumatic EO
Klebsiella	Bacterial endogenous EO
Candida	Fungal endogenous EO

Symptoms :

- Pain.
- Redness.
- Loss of vision.

Signs :

- Anterior chamber :



- Posterior segment :
  - Yellowish exudates in vitreous.

Management :

Assessment of vision

- Clinical blindness (No perception of light) :
  - Surgical removal : Evisceration (Removal of all layers of eyeball except sclera).
- Visual potential present
  - Posterior segment coverage + Anterior segment coverage + Pars plana Vitrectomy
  - Posterior segment coverage → Intravitreal antibiotics :
    - Vancomycin : 1 mg in 0.1 mL
    - Ceftazidime : 2.2 mg in 0.1 mL
  - Anterior segment coverage → Topical antibiotics

Note :

Systemic antibiotics have no role in healing endophthalmitis.

Flat/shallow AC :

- Wound leak.
- Malignant glaucoma.
- Pupillary block glaucoma.
- Cilio-choroidal detachment

Other complications :

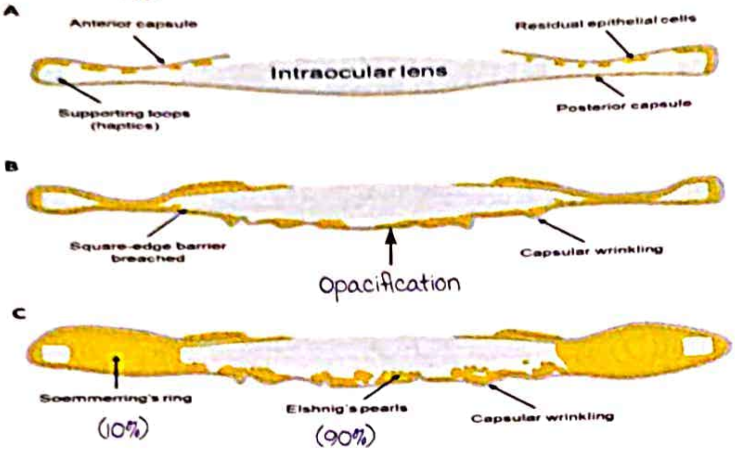
- Uveitis.
- Retinal detachment.
- Astigmatism.

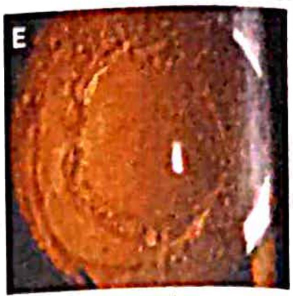
00:34:40

Late Post Operative Complications

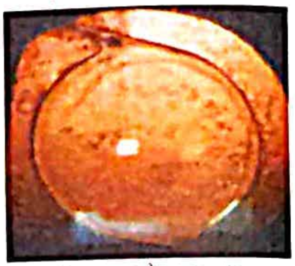
POSTERIOR CAPSULAR OPACIFICATION (PCO)

- AKA 2° cataract, after cataract.
- m/c late complication (6-12 weeks).
- Causes gradual painless loss of vision.
- Square edged IOL reduce the occurrence of PCO.
- Pathology : Posterior collection of residual epithelial cells.


A
Anterior capsule
Residual epithelial cells
Intraocular lens
Supporting loops (haptics)
Posterior capsule
B
Square-edge barrier breached
Capsular wrinkling
Opacification
C
Soemmering's ring
(10%)
Elshnig's pearls
(90%)
Capsular wrinkling



Nd : YAG laser post capsulotomy



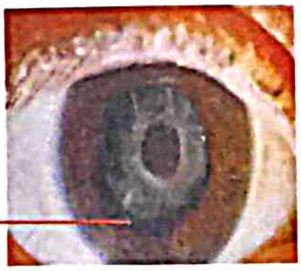
Elschnig's pearls

Treatment :

- Nd : YAG laser posterior capsulotomy.
  - mechanism : Photodisruption.

ANTERIOR CAPSULE PHIMOSIS

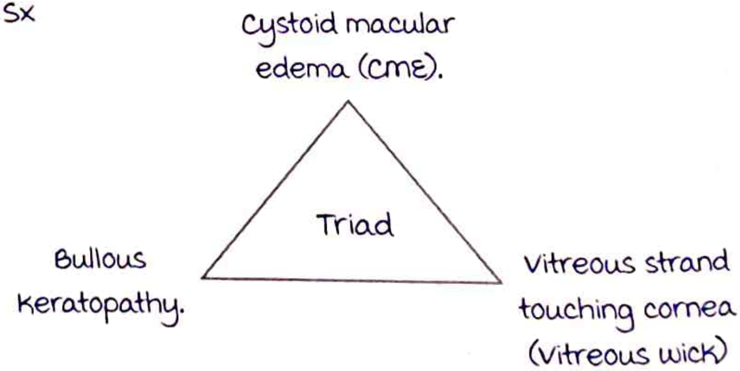
Contraction of anterior capsule d/t fibrosis → IOL decentration



AC phimosis

IRVIN GLASS SYNDROME

Occurs 6 to 10 weeks after Sx


Cystoid macular edema (CME).
Triad
Bullous Keratopathy.
Vitreous strand touching cornea (Vitreous wick)

IOL DISLOCATION

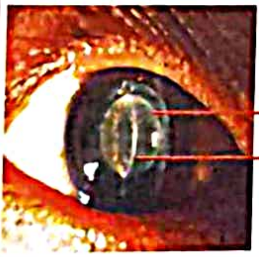
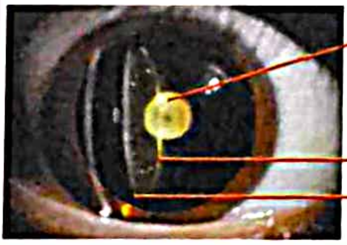
- Upwards : Sunrise syndrome.
- Downwards : Sunset syndrome.
- Lost lens syndrome : IOL falls into vitreous.
  - D/t PCR.
  - Spontaneous : Hallerman-Steriff syndrome.

CONGENITAL CATARACT, ECTOPIA LENTIS AND MISCELLANEOUS

Congenital Cataract

00:00:07

CLASSIFICATION

	Infantile Primary Congenital Cataract	Developmental Cataract
Age of onset	At birth (no progression)	
Presentation	Within 1 year	> 1 year
Symptoms	Severe loss of vision : - D/t interference with foveal fixation. - Foveal fixation : Brain ascertains that the best image is formed on retina at foveal point by 3-4 months of age. - Failure of fixation : Sensory deprivation amblyopia (without any organic cause).	minimal loss of vision
management	Surgery upon diagnosis (within 12 weeks of age)	Wait & watch
Types	Lamellar/Zonular cataract (m/c) : - Autosomal dominant inheritance. - Associated with a. Congenital rubella syndrome b. Vitamin D deficiency	• Blue-dot cataract (m/c) - Associated with Down syndrome . • Posterior polar cataract : - Associated with : a. mittendorf dots : Remnants of hyaloid artery /Tunica vasculosa lentis. b. PHPV (persistent hyperplastic primary vitreous).
Appearance	 Spoke-like opacities : Riders Opacity in fetal nucleus	 Onion ring/whirled whorled appearance Posterior capsule Anterior capsule

TREATMENT

Lens aspiration (d/t soft Lens) + Posterior capsulorhexis (to avoid PSCC in the future) + Anterior vitrectomy (to avoid mixing of aqueous/vitreous)

↓ usually > 2y of age → If done < 2 yr of age:

IOL implantation :

IOL undercorrection required as power of lens increases with age (Power at Birth: 2-3D → Hypermetropia) :
- <2yrs : undercorrection by 20%.
- 2-8 yr : undercorrection by 10%.

If done < 2 yr of age: small IOL implantation may lead to lens dislocation as size of eyeball increases with age

----- Active space -----

Note : Congenital rubella syndrome (CRS)

- Occurrence : 1st trimester of pregnancy.
- Ocular manifestation :
  a. Nuclear pearly cataract (m/c cataract in CRS).
  b. micro-ophthalmus.
  c. Salt & pepper retinopathy.

Axial length of eyeball

- At birth : 17mm
- ~7 yr of age : 24mm (adult size)

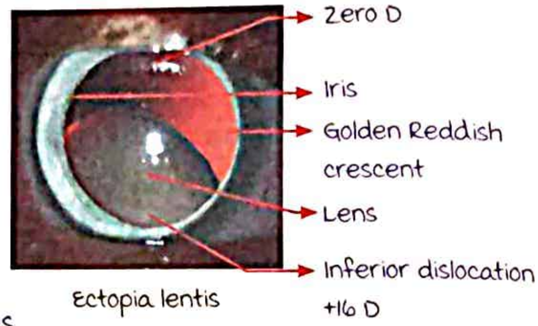
1. Axial length of eyeball
2. Congenital Rubella Syndrome

Other Diseases of Lens

00:21:50

ECTOPIA LENTIS

- Displacement of lens
  - Partial dislocation (subluxation).
  - Complete dislocation.
- monocular diplopia may occur.



Ectopia lentis

Causes :

a. Blunt trauma (m/c) : d/t break in zonules.

b. Familial : Autosomal dominant inheritance.

c. Ectopia lentis et pupillae : Congenital bilateral condition where lens & pupil move in opposite directions.

d. Aniridia : Absence of Iris.

e. marfan's syndrome : m/c systemic association.
  - Superotemporal ectopia lentis.
  - Cornea plana & megalocornea (flat & large cornea).
  - Lattice degeneration of retina. } D/t ↑ axial length of eye
  - Axial myopia. }
  - Dilator pupillae hypoplasia.

f. Homocystinuria :
  - Deficiency of cysteine → Accumulation of homocysteine & methionine → Zonular disintegration → ectopia lentis.
  - Inferonasal ectopia lentis.
  - Diagnosis : Cyanide nitroprusside test.

g. Weill-marchesani syndrome :
  - Forward / Anterior ectopia lentis.
  - Short stature.
  - Small, stubby fingers.

h. Sulfite oxidase deficiency.

i. Hyperlysinemia.

j. Ehlers-Danlos syndrome.

management :

a. Partial Subluxation : Spectacles.

b. Clinically significant Subluxation : Surgery.

- Refixate the lens.
- ECCE/ICCE + IOL implantation.

LENTICONUS

Cone-shaped lens.

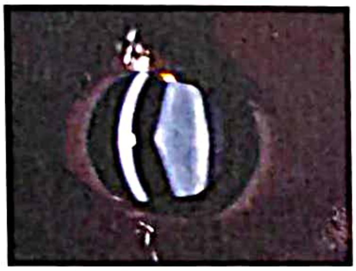
Types :

Anterior lenticonus :

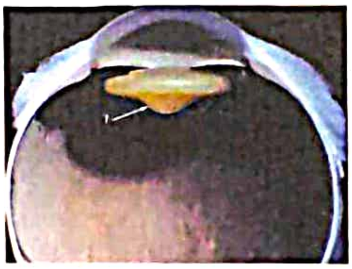
- Associated with (mnemonic : SAW)
  - Spina Bifida
  - Alport syndrome (SNHL + abnormal glomerular BM)
  - Wardenburg syndrome

Posterior lenticonus :

- Associated with :
  - Lowe syndrome (AKA oculorenal syndrome)
  - X-linked inheritance
- Features :
  - Congenital cataract
  - Congenital glaucoma
  - microphakia



Anterior lenticonus



Posterior lenticonus

MICROSPHEROPHAKIA

- Congenital defect.
- Lens → Small → ↑curvature → myopia
  → Spherical → dislocation → 2° Angle-closure glaucoma.
- Association : marfan's, Weil marchesani syndrome.

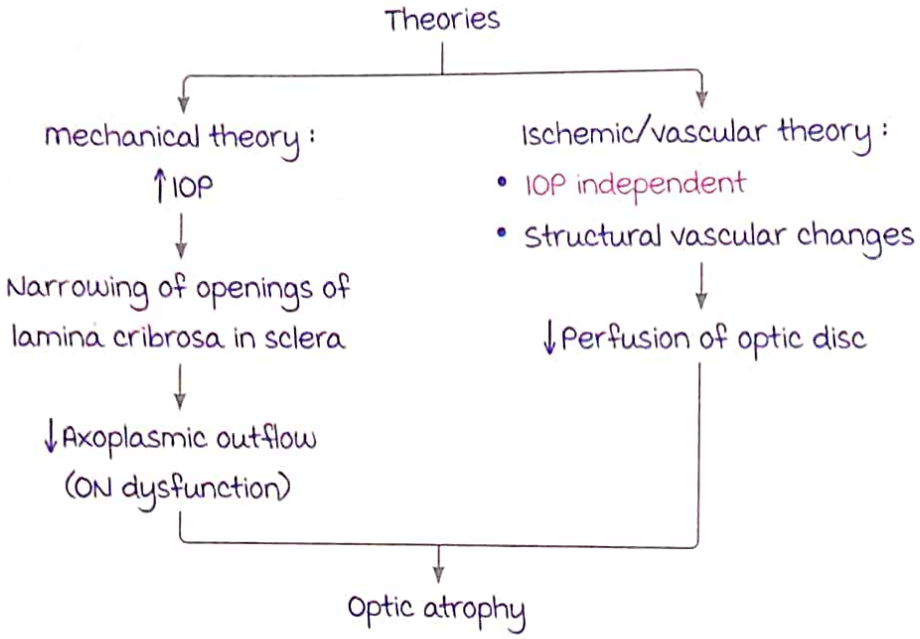
GLAUCOMA : WHAT AND HOW?

Overview of Glaucoma

00:01:50

Defining features	Investigations
1. Progressive optic neuropathy with characteristic optic disc (visible part of ON) changes.	Examination of optic disc : Ophthalmoscopy.
2. Irreversible visual field changes : • Drugs ⟶ Only prevent progression.	Visual field examination : Perimetry.
3. Intra ocular pressure (IOP) may/may not be raised (IOP ⟶ Risk factor for glaucoma).	measure IOP : Tonometry.

Pathophysiology :


Theories
mechanical theory :
↑IOP
Narrowing of openings of lamina cribrosa in sclera
↓Axoplasmic outflow (ON dysfunction)
Ischemic/vascular theory :
• IOP independent
• Structural vascular changes
↓Perfusion of optic disc
Optic atrophy

Aqueous Humour

00:09:20

Contributes to IOP.

NORMAL FLOW

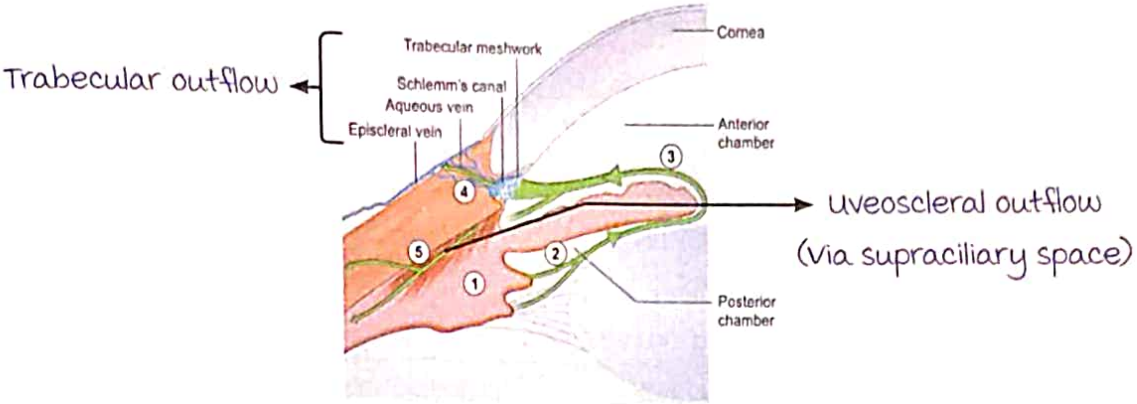
Production : Non pigmented epithelium of ciliary processes. (1)

↓

Secretion : Into posterior chamber (PC). (2)

Secretion : Into posterior chamber (PC) (2)

Pathway
- ↓ via pupil
- Reaches anterior chamber (AC) (3)
- ↓
- Angle of anterior chamber (Irido corneal angle) (4) & (5)


Trabecular outflow
Trabecular meshwork
Schlemm's canal
Aqueous vein
Episcleral vein
Cornea
Anterior chamber
③
④
⑤
②
①
Uveoscleral outflow
(Via supraciliary space)
Posterior chamber

Aqueous outflow

Outflow

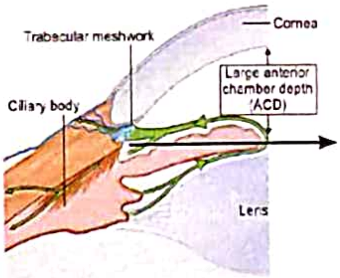
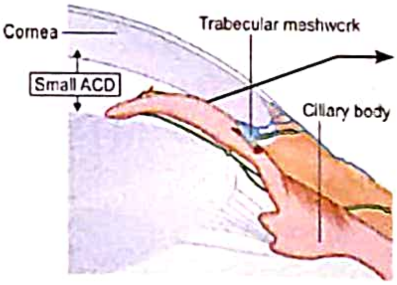
Trabecular (90%) :
- Pressure dependent
- Pathway : AC
  ↓
  Trabecular meshwork
  ↓
  Schlemm's canal
  ↓
  Aqueous vein
  ↓
  Episcleral vein

Uveoscleral (10%) :
- Pressure independent
- Pathway : AC
  ↓
  Supraciliary space

Open Angle Glaucoma & Angle Closure Glaucoma

00:18:55

Types of Glaucoma (G) :

	Open Angle Glaucoma	Angle Closure Glaucoma
Pathogenesis	 Trabecular meshwork Cornea Ciliary body Large anterior chamber depth (ACD) Lens Blockage of trabecular meshwork	 Cornea Trabecular meshwork Small ACD Ciliary body Iris in apposition to cornea ↓ Trabecular meshwork ↓ Blocked Glaucoma

	Open Angle Glaucoma	Angle Closure Glaucoma
Rx	Trabeculectomy	Iridectomy
Gonioscopy : Examination of angle of the anterior chamber	Angle open	Angle closed

Mechanism and Causes :

Open angle :
When the anterior chamber angle structures are visible..

Angle closure :
When the iris is in apposition to cornea → Thus, angle structures are obscure.

Open angle
Depending on site of blockage

Pre-trabecular :
membrane covering the angle.
↓
- Neovascular G
- PPMD
- Iridocorneal endothelial syndrome (ICE)
- Fuchs' heterochromic iridocyclitis
- Trauma

Trabecular :
Blockage of TM.
↓
- POAG
- Phacolytic G
- Pigmentary G
- Ghost cell G
- Pseudoexfoliative G
- Uveitis
- Trauma

Post-trabecular :
Obstruction/collapse of Schlemm's canal (or) Elevated episcleral venous pressure
↓
- Sickle cell anemia
- Elevated episcleral pressure
- CCF
- Superior vena cava syndrome
- Thyrotoxicosis

Angle closure

Pulling :
membrane covering the angle.
↓
Contracts
↓
Pulls peripheral iris towards cornea
↓
- Neovascular G
- Iridocorneal endothelial syndrome (ICE)
- Trauma

Pushing :

Apposition b/w lens and pupillary margins
↓
With pupillary block :
- PACG
- Phacomorphic G
- Phacotopic G
- Aphakia
- Pseudophakia

Without pupillary block :
- Plateau iris
- Malignant G
- Tumors
- Post surgical

Investigations :

Optic disc examination :

- Direct ophthalmoscopy.
- Slit lamp biomicroscopy.
- Confocal Scanning Laser Tomography (CSLO).
- Spectral domain OCT.
- Confocal Scanning Laser Polarimetry (SLP).

Visual field examination :

Perimetry

- Kinetic
  E.g. Goldmann perimeter, Bjerrum tangent screen
- Static
  E.g. Humphrey field analyzer (HFA), Dicon, Octopus

IOP measurement :

Tonometry :

- Indentation tonometry.
- Non-contact tonometry.
- Transpalpebral tonometry.
- Applanation tonometry.

Applanation tonometry

- Fixed force :
  1. Maklakov
  2. Tonomat
  3. Barraquer
- Fixed area :
  1. Goldmann's
  2. Perkin's
  3. Pneumatic
  4. Draeger
  5. mackay marg
  6. Tonopen
  7. PASCAL

Examination of angle of anterior chamber :

- Van Herrick's method.
- Oblique Flashlight Test.
- Gonioscopy.
- Ultrasonic Biomicroscopy (UBM).
- Anterior segment – ASOCT.

INVESTIGATIONS FOR GLAUCOMA

00:00:10

Intra Ocular Pressure (IOP) Measurement : Tonometry

NORMAL IOP

Normal pressure = 11-21 mmHg.

Diurnal variation (maximum) : 8 am - 12 pm (mean : 10 pm)

IOP measurement of Corneal Thickness :

- Thin cornea : False low IOP.
  - Post refractive Sx cases.
- Thick cornea : False high IOP.

TYPES OF TONOMETRY

1. Digital Tonometry :

- By palpation.
- Performed in downgaze (palpation on sclera).
- Tonometry of choice in keratoprosthesis patients.

Digital Tonometry

Findings :

Soft to Touch : Low IOP

Firm to touch : Normal IOP

Hard to touch : High IOP

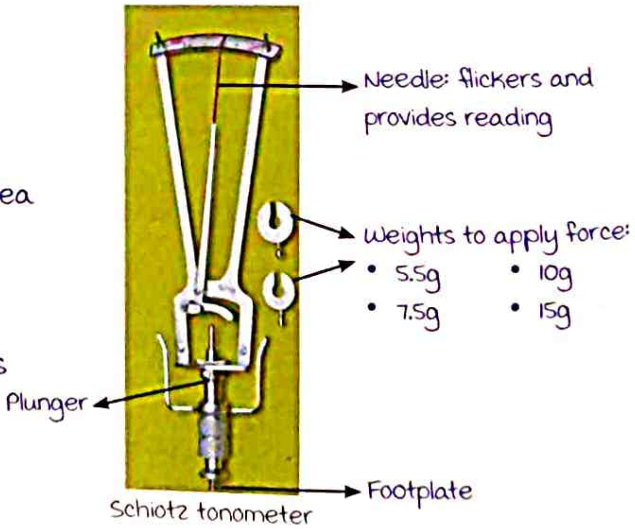
2. Indentation Tonometry :

Principle of displacement of fluid used to calculate IOP.

a) Schiotz tonometer :

- Hand held instrument.
- Patient supine.
- Footplate → Contact with cornea

Pressure applied
↓
Displaces aqueous
↓
measures IOP


Needle: flickers and provides reading
Weights to apply force:
• 5.5g • 10g
• 7.5g • 15g
Plunger
Footplate

Schiotz tonometer

- Friedenwald's Normogram : chart to determine IOP

parameters

Ocular rigidity

Weight used

Needle reading

b) Rebound Tonometry :

Instrument of choice for self-monitoring of IOP at home.

eg. long term glaucoma patient.

- Drawbacks :

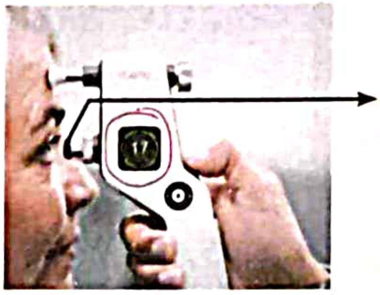
↑ Interobserver and intraobserver variability in reading

↓

↑ chances of inaccurate measurement of IOP

↓

WHO recommended against use



Rebound Tonometry

3. Applanation Tonometry (AT) :

- Principle : Imbert Fick law → $P = F/A$

P : Pressure in contained sphere (eyeball).

F : Force applied

A : Area applanated/pressed

- Types :

Fixed Force (not preferred) :

Eg :

- Maklakov tonometer.
- Barraquer tonometer.
- Tonomat.

Fixed area :

Eg :

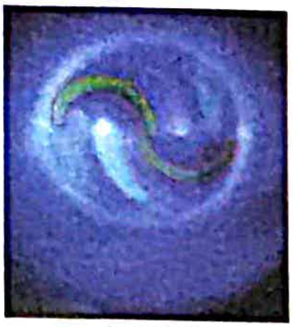
- Goldmann's AT.
- Perkins AT.
- Mackay marg.
- Tonopen.
- Pneumatic Tonometer.
- Pascal Tonometer.

a) Goldmann's AT :

- Gold standard tonometer.
- Slit lamp mounted instrument.
- Area = $3.06\ mm^2$.
- Parts :
    - Body → contains knob to alter the force applied on cornea → to determine eye pressure.
    - Neck (long and slender).
    - Conical tip.
    - Biprism → touches cornea.

----- Active space -----

Cobalt blue light



AT rings

Perkins Tonometer

- Procedure :

Step 1 : Topical anesthesia → Abolish corneal reflex
  - Drug : Proparacaine eye drops.
  - Lower lid retracted → 1 drop administered in inferior fornix.

Step 2 : Stain cornea with fluorescein sodium ophthalmic strip (orange strip) :
  - Lower lid retracted → Stain lower palpebral conjunctiva & lower fornix.
  - Blink 4 - 5 times → dye spread throughout cornea.

Step 3 : Illuminate cornea under cobalt blue light → green fluorescence produced.

Step 4 : Tip of tonometer advanced towards cornea → makes contact

↓

Biprism in tip splits light into 2 mires (Hemispheres).

Adjust force applied (knobs)

↓

Inner margin of both AT rings/mires touch each other

b. Perkins AT : Preferred choice in :
- Children
- Bed ridden
- Anaesthetized patients

c. mackay - marg AT :
Preferred choices in irregular edematous scarred corneas.

d. Pneumatic AT/Pneumotonometer
Choice for continuous IOP measurement.

e. Pascal AT/dynamic contour tonometry (DCT) :
- Influenced by changes in corneal curvature.
- Not influenced by changes in corneal thickness.

↓

- Investigation of choice following refractive surgeries like LASIK.

4. Non contact tonometry :
- AKA pulsair/airpuff tonometry.
- Preferred choice for screening of IOP in OPD camps.
  - Quick method
  - No contact with eye → sterilization not required (cross infection risk).
- method : Puff of air exerted → compresses cornea → measures IOP.

5. Transpalpebral tonometry :
- Not commonly used.
- measuring IOP through eyelid
- Preferred in case of corneal pathology.
- Instrument :
  - Diaton Tonometer.
  - Proview Tonometer.

Transpalpebral tonometry.

Visual Field Examination : Perimetry

00:34:10

----- Active space -----

KINETIC PERIMETRY

- Target moves
- monocular testing of visual field.

E.g. :

- Goldmann perimetry.
- Bjerrum's tangent screen.

STATIC PERIMETRY

Target is stationary.

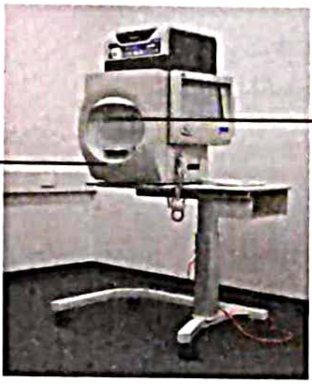
Types

Supra Threshold :
used for screening

Threshold :

- used for monitoring of glaucoma
- E.g., a. Humphrey Field Analyzer (HFA) : m/c used.
  b. Octopus Perimeter.

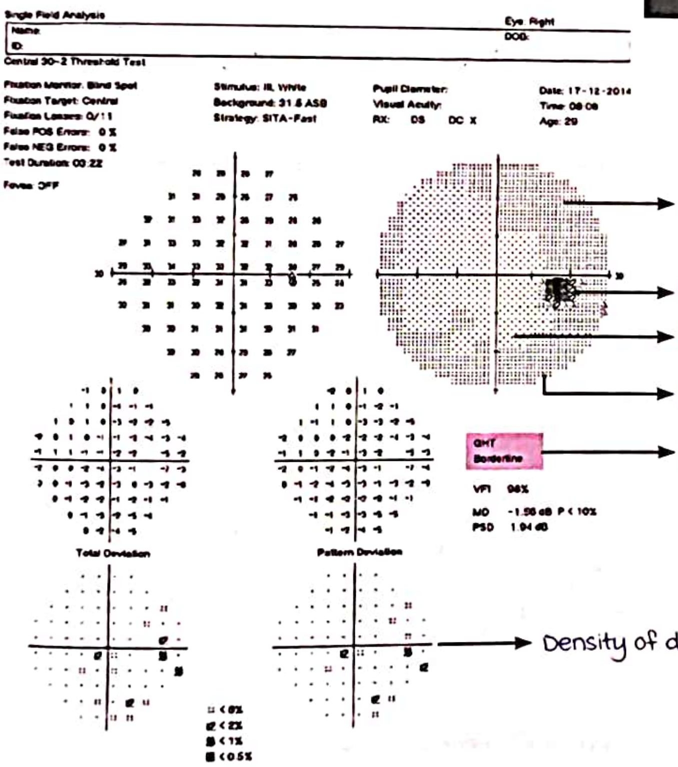
Humphrey Field Analyzer (HFA) :



Apparatus

Patient chin rest

Curved screen containing white LEDs at different points on a white background
↓
Patient clicks a remote when a particular LED is visible
↓
Findings are analyzed



Single Field Analysis
Name:
ID:
Eye: Right
DOB:
Central 30-2 Threshold Test

Fixation Monitor: Blind Spot
Fixation Target: Central
Fixation Losses: 0/11
False POS Errors: 0 %
False NEG Errors: 0 %
Test Duration: 03:22
Fovea: OFF

Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA-Fast

Pupil Diameter:
Visual Acuity:
RX: DS DC X

Date: 17-12-2014
Time: 08:08
Age: 29

GHT
Borderline

VFI 98%
MD -1.58 dB P < 10%
PSD 1.94 dB

VF

Blind spot

Sparse dots : VF area maybe defective

↑ Density of dots → better vision

GHT : Glaucoma Hemifield Test

Density of dots represented

Findings

Testing patterns :

a. 24-2 :
- Central 24° VF tested.
- 2 points separated by 6°.

b. 30-2 :
- Central 30° VF tested.
- 2 points separated by 6°.

c. 10-2 :
- Central 10° VF tested.

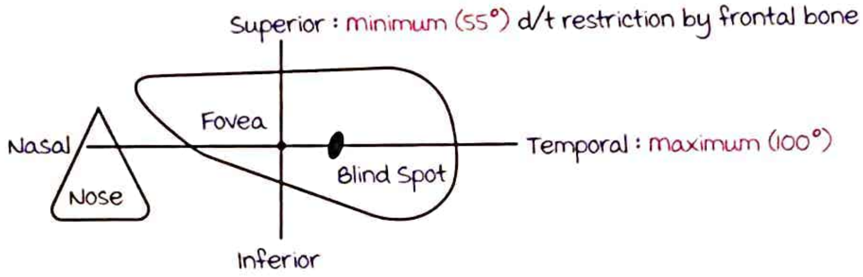
d. Full field – 120 point testing :
- Central & peripheral VF examined. Eg: Bitemporal heminopia → requires examination of temporal most (peripheral) and central VF.

Other Testing Strategies :

- SITA (Swedish Interactive Threshold Algorithm).
- SWAP :
    - Blue on yellow stimuli used.
    - Used for examination of Koniocellular pathway.
- FDT :
    - Black and white flickering stimuli used.
    - Used for examination of magnocellular pathway.
- Ring perimetry :
    - Tests peripheral vision.
    - Used for examination of parvocellular pathway.

NORMAL VISUAL FIELD

Shape : Horizontally oval with inferonasal notch.


Superior : minimum (55°) d/t restriction by frontal bone
Nasal
Fovea
Temporal : maximum (100°)
Nose
Blind Spot
Inferior

Optic Disc (OD) Examination

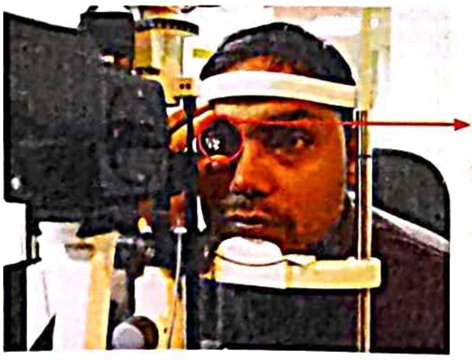
00:51:22

1. Ophthalmoscopy :

Types
- Direct : Used for OD examination
- Indirect

2. Slit lamp biomicroscopy :

Slit lamp



Examination with an external lens

Hand held external lens, power can be :
- + 90D convex lens (m/c).
- + 78D convex lens.

----- Active space -----

3. Other tests :

- Confocal scanning laser tomography.
- Spectral domain OCT.
- Confocal scanning laser polarimetry (SLP).

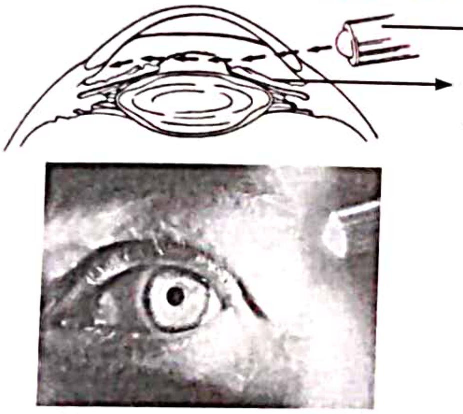
Examination of Angle of Anterior Chamber

00:56:23

OBLIQUE FLASHLIGHT METHOD

Eye illuminated temporally (obliquely)
↓
Temporal iris illuminated
↓
Findings can be :

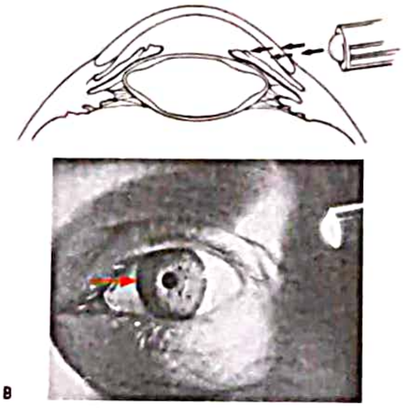
Nasal iris illuminated



- AC deep, open
- Iris flat

↓
Entire iris illuminated
↓
Indicative of OAG

Shadow over nasal iris



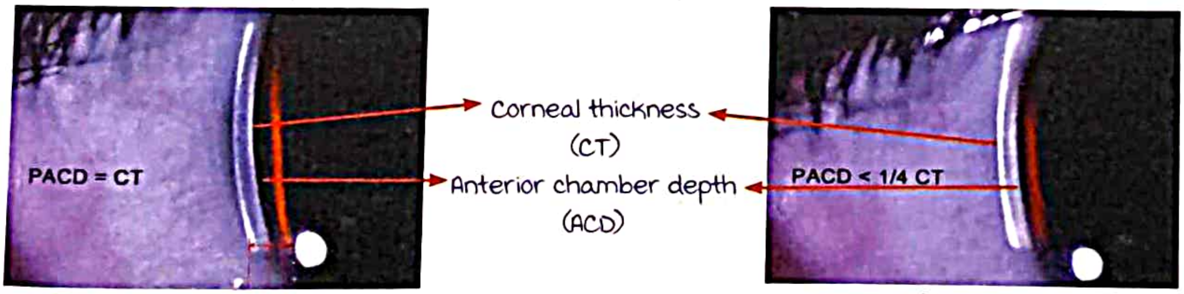
- Iris moved forward shallow AC

↓
Indicative of possible ACG

Eclipse sign : shadow over nasal iris

VAN HERICK'S METHOD (SLIT LAMP BASED INVESTIGATION)

Compare PACD (Peripheral anterior chamber depth) to CT (Corneal thickness).



ACD = CT
Open angle (Grade 4)

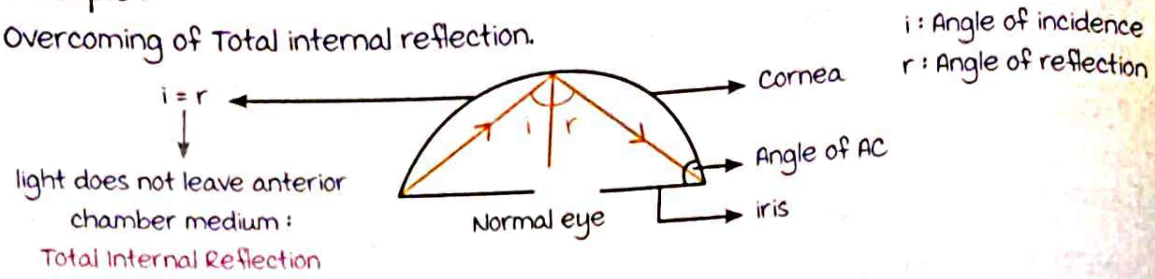
ACD < CT
Angle closed (Grade I)

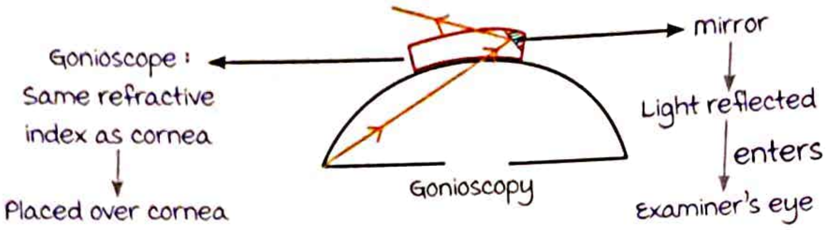
GONIOSCOPY

Visualize structures in angle of AC.

Principle :

Overcoming of Total internal reflection.


i : Angle of incidence
r : Angle of reflection
i = r
light does not leave anterior chamber medium :
Total Internal Reflection
i
r
Cornea
Angle of AC
iris
Normal eye


Gonioscope :
Same refractive index as cornea
Placed over cornea
mirror
Light reflected
enters
Examiner's eye
Gonioscopy

- Gonioscopy contraindicated in dilated pupil (Dilated pupil → iris tissue collected near AC angle → structures of angle obscured).

Types

Direct : using goniolens

Eg : Koeppe Lens
- Swan Jacob lens
- Barkan Lens

- Used in :
  - Children
  - Infants
  - Bedridden patients

Indirect : using gonioprism
- Sitting positioning
- Slit lamp based

Eg : - Zeiss 4 mirror gonioscope :
- Posner 4 mirror gonioscope
- Goldmann's 3 mirror gonioscope

} contains → Superior, Inferior, nasal, temporal mirrors ↓ All quadrants of angle of AC can be examined

Structures in the angle of AC

Mnemonic : Can't See This Stuff.

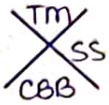
Posterior ↓ Anterior
- Ciliary body band (CBB)
- Scleral Spur (SS)
- Trabecular meshwork (TM)
- Schwalbe's line : Peripheral termination of descemet's membrane

} As iris moves forward ↓ structures of AC serially get obscured

Grading of Gonioscopy findings :

Scheie's grading :

- m/c grading.
- Based on visible angle structures :
  - Grade I : All structures visible (wide open).
  - Grade II : CBB obscured.
  - Grade III : CB, SS, posterior TM obscured.
  - Grade IV : Only schwalbe line visible (closed).
- Schematic Representation :



Shaffer Grading :

- Based on angular width :
  - Grade I : <10° (closed).
  - Grade II : 10°-20°.
  - Grade III : 25°-35°.
  - Grade IV : 35°-45° (wide open).

Spaeth grading :

Based on :

- Angular width.
- Configuration of peripheral iris.
- Iris root configuration.

Examination Overview

01:17:20

Optic disc examination :	Visual field examination perimetry :	IOP measurement Tonometry :	Examination of angle of anterior chamber :
• Direct Ophthalmoscopy • Slit Lamp Biomicroscopy • Confocal Scanning Laser Tomography (CSLO) • Spectral Domain OCT • Confocal Scanning Laser Polarimetry (SLP)	Kinetic • E.g. Goldmann perimeter, Bjerrum tangent screen Static • E.g. Humphrey field analyzer (HFA), Dicon, octopus	• Indentation Tonometry • Non-contact Tonometry • Transpalpebral Tonometry • Applanation Tonometry Fixed force - Maklakov - Tonomat - Barraquer Fixed Area - Goldmann's - Perkins - Pneumatic - Draeger - Mackay marg - Tonopen - PASCAL	• Van Herick's method • Oblique Flashlight Test • Gonioscopy • Ultrasonic Biomicroscopy (UBM) • Anterior Segment - ASOCT

----- Active space -----

PRIMARY OPEN ANGLE GLAUCOMA

Types of Glaucoma :

- Acquired (In adults) :
  - Primary :
    - 1° Open Angle Glaucoma (POAG)
    - 1° Angle Closure Glaucoma (PACG)
  - Secondary :
    - PXFG
    - Pigmentary glaucoma
    - ICE syndrome
    - Malignant glaucoma
    - Lens induced glaucoma
- Congenital (In children) :
  - Primary :
    - D/t birth malformations
  - Secondary :
    - Associated with other congenital disease
    - AKA developmental glaucoma

POAG/Chronic Simple Glaucoma

00:02:10

OVERVIEW

Chronic disease.

- Symptoms : Minimal.
- Signs : Detectable.

M/C with age > 50 yrs.

Can be hereditary.

- Contributory genes :
  - Optineurin.
  - WDR-36.
  - MYOC (myocilin gene) → Also implicated in cystic fibrosis.

Mechanism :

Blockage of TM (Trabecular meshwork)
→ Obstruction of outflow.

Symptoms :

1. Headache.
2. Difficulty/delay in dark adaptation.
3. Frequent changes in presbyopic glasses.

} Non diagnostic

Note : Frequent change in glasses seen in cataract d/t change in refractory index of lens.

Signs of POAG

00:12:05

----- Active space -----

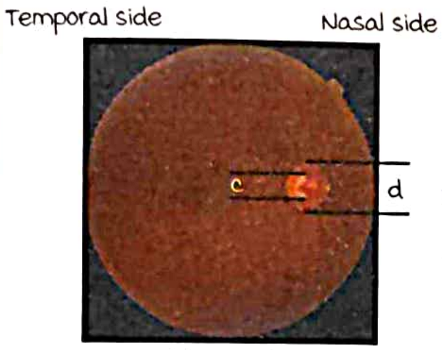
1. IOP (Intra Ocular Pressure) :

- > 21 mmHg (Normal range : 11-21 mmHg) or
- > 5 mmHg difference b/w both eyes or
- > 8 mmHg difference in diurnal variation.

Normal Tension Glaucoma (NTG) :

- If OD changes + VF changes + Normal IOP.
- Commonly a/w :
  - Raynaud phenomena.
  - migraine.
  - ↓ blood flow velocity in ophthalmic artery.

2. OD changes :

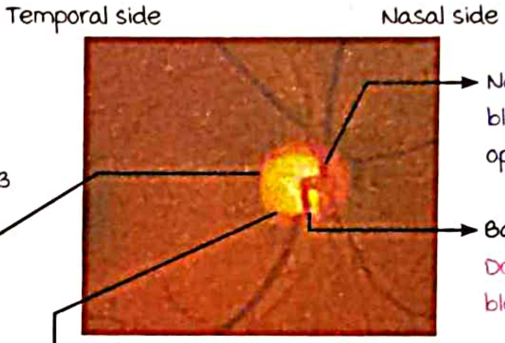


Normal OD

Normal c/d ratio = 0.3

Vertical cupping :

- c/d ratio > 0.5
- Loss of NRR (Neuroretinal rim).



Glaucomatous optic atrophy

Nasalization of blood vessels within optic cup.

Bayonetting sign : Double angulation of blood vessels.

Loss of NRR : Inferotemporal part lost earliest → Polar Notching.

Laminar dot sign : Fenestrations in sclera (Lamina cribrosa) become visible.
↓
Dotted appearance at base of OC.

Peri-papillary atrophy : Atrophy around OD on temporal side.

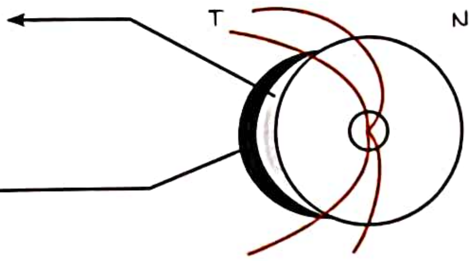
Splinter hemorrhages at OD.

β zone/Temporal myopic crescent :

- Not specific for OAG.
- Acts as confounding factor.

α zone :

Specific for OAG.

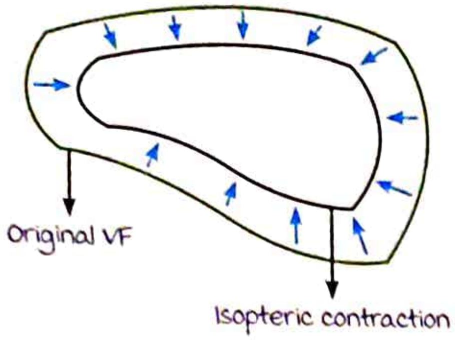


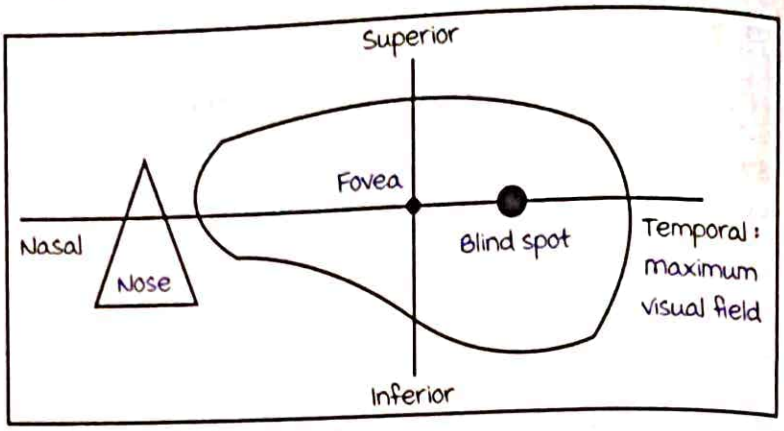
Peripapillary atrophy

3. Visual field (VF) changes :

Irreversible.

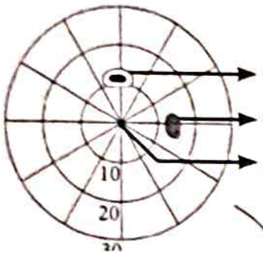
a. Earliest VF change : Isopteric contraction (Entire VF contraction).


Original VF
Isopteric contraction


Superior
Fovea
Nasal
Nose
Blind spot
Temporal : maximum visual field
Inferior

Note : Isopter → 2 Dimensional plot of 3D visual field

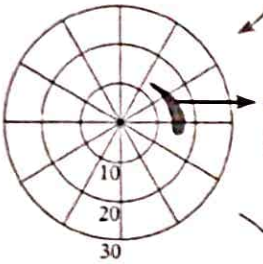
b. Baring of blind spot.



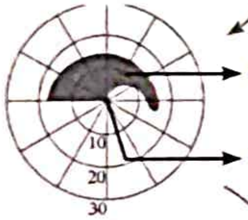
c. Paracentral scotoma → Earliest clinically significant VFD.
→ Central 10° of VF intact.

Blind spot

Fovea



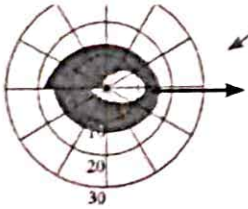
d. Seidel's scotoma : Sickle shaped/comma shaped (Fusion of paracentral scotoma with blind spot)



e. Arcuate/Bjerrum scotoma : Altitudinal visual field defect (Respects horizontal midline).

Fovea spared

Inferior arcuate fibres destroyed



f. Double Arcuate scotoma/Ring scotoma

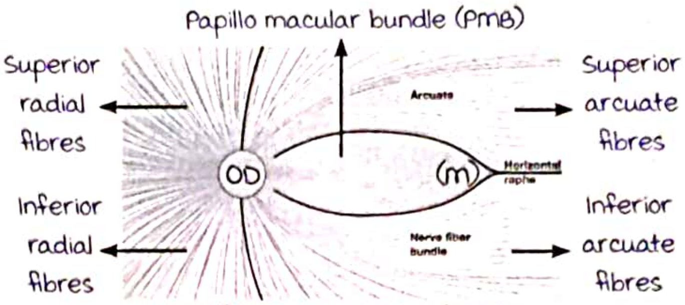
- Arcuate fibres : First to be affected in OAG.
- Damage contained in Bjerrum's area : Area of VF containing arcuate fibres.

c. Nasal step defects :
- Occur d/t unequal loss of nerve fibres/VF in upper & lower hemisphere.
- 2 types :
  - Peripheral.
  - Central : Roenne's.

d. Vertical septal defects

e. Loss of Central field

f. Loss of Temporal island of vision.
- Temporal island :
  - most resistant part of VF.
  - Last part to be affected.


Papillo macular bundle (PMB)
Superior radial fibres
Inferior radial fibres
Superior arcuate fibres
Inferior arcuate fibres
Arcuate
OD
(m)
Horizontal raphe
Nerve fiber bundle

Nerve fibres arranged in fundus :

When damage to arcuate fibres occurs
↓
PMB spread
↓
Arc shaped defect in VF.

m : macula
OD : Optic disc

Medical Mx : Topical Eye Drops

00:43:15

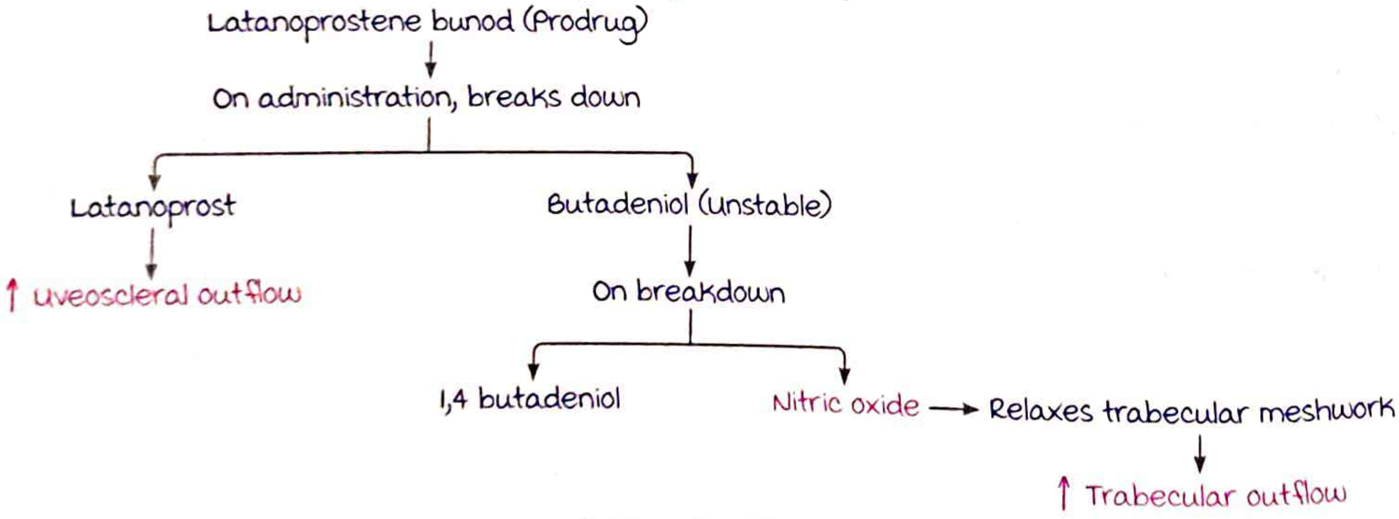
Lifelong Rx :

Drugs	MOA	Side effects
a. Prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) analogues : Drugs of choice : • Latanoprost • Bimatoprost • Travoprost • Tafluprost } OD : Good compliance	↑ Uveoscleral outflow	• Hyperpigmentation of iris • Hypertrichosis (↑ growth of eyelashes) – Bimatoprost : FDA approved for hypotrichosis. • Uveitis. • Cystoid macular edema. • Reactivation of Herpes Keratitis : – Take h/o previous corneal/ herpetic infections. • Iris cysts.
b. β blockers : • Timolol : Non selective β blocker > • Betaxolol : Selective β blocker } BD	β receptor blocking ↓ ↓ Aqueous production	• Blepharoconjunctivitis : Inflammation of conjunctiva and eyelid margin. • Naso lacrimal duct (NLD) blockage. • Corneal anesthesia on continuous use (Especially Timolol). • Aphakic cystoid macular edema – Specific to Betaxolol.

----- Active space -----

Drugs	MOA	Side effects
c. Carbonic Anhydrase Inhibitors (CAIs) : • Acetazolamide – Only available orally. • Brinzolamide • Dorzolamide – Rx of childhood glaucoma.	Inhibit CA II isoform on sphincter pupillae & ciliary muscle ↓ ↓ Aqueous production	Can cause hypersensitivity in patients with sulfa allergy.
d. Rho Kinase Inhibitors : Netarsudil : • Dose : 0.02% OD	Dual action drug : 1. ↓ Aqueous production. 2. ↑ Trabecular outflow by : • Promoting actin-myosin contraction. • ↓ Episcleral venous pressure.	• Congestion/hyperemia. • Corneal verticillata (Spindle shaped deposits). • Subconjunctival haemorrhage.
e. α-agonists i. Apraclonidine : • more preferred agent for glaucoma. • DOC for post laser rise in IOP.	↓ Aqueous production + ↑ Trabecular outflow	• Eyelid retraction. • mydriasis (D/t $\alpha_2 > \alpha_1$ agonism). • Follicular conjunctivitis.
ii. Brimonidine : 2nd line agent for glaucoma.	↓ Aqueous production + ↑ Uveoscleral outflow	CNS suppression (D/t BBB crossing) • Drowsiness • Depression • Apnea • Bradycardia } C/I in children
iii. Dipivefrin : Not used	-	Black conjunctival pigmentation

f. Nitric oxide donating prostaglandin analog :


Latanoprostene bunod (Prodrug)
On administration, breaks down
Latanoprost
↑ Uveoscleral outflow
Butadeniol (Unstable)
On breakdown
1,4 butadeniol
Nitric oxide
Relaxes trabecular meshwork
↑ Trabecular outflow

Note :
Pilocarpine → Constricts pupil → Not useful in POAG.
mannitol → Emergency drug → Not useful for chronic symptoms.

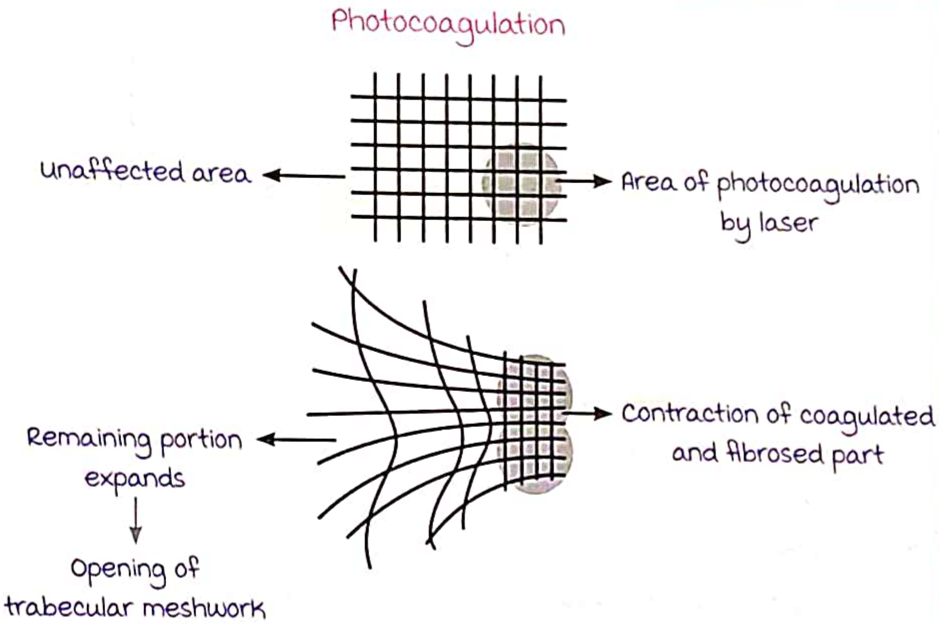
Surgical Mx of POAG

01:10:40

when medical Rx fails.

CLOSED SURGERY

Laser trabeculoplasty (LT) :

	Argon LT	SLT (Selective LT)
wavelength used	512 nm	532 nm : Frequency doubled Nd-YAG laser.
mechanism	 Photocoagulation unaffected area Area of photocoagulation by laser Remaining portion expands Opening of trabecular meshwork Contraction of coagulated and fibrosed part	
Remarks	-	TOC for prophylaxis of OAG in other eye (OAG is bilateral)

OPEN SURGERY

If not controlled by closed Sx.

Penetrating surgery - Trabeculectomy :
Creating fistula b/w AC and subtenon space → Aqueous drainage.

Non-penetrating Sx :

- Deep sclerectomy.
- Viscocanalostomy.

----- Active space -----

SURGERY WITH IMPLANTS

Done for resistant glaucoma.

Seton Sx - Bypass :

- Implant a glaucoma drainage device.
- Parts :

1. Tube → Inserted into AC (Sub-tenon or subconjunctivally) → Enables outflow.
2. Plate → Positioned in sub-tenon space → Collects fluid

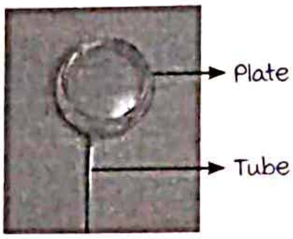
Types of drainage device :

Non-valved :
Continuous outflow

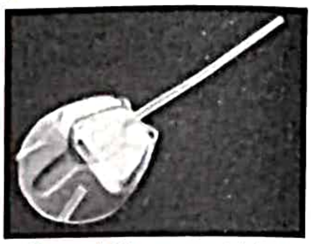
Valved :
Stops outflow when required

Express mini shunt
made of stainless steel

Baerveldt device



malteno device



Ahmed Glaucoma Valve
(AGV)

MIGS (Minimally Invasive Glaucoma Sx) :

Stents through TM → Enables outflow.

Advantages :

- Small incision.
- ↓ Chance of post operation infection (Eg. : Endophthalmitis).

Types :

Procedure	Site of Insertion	Material	Indication
iStent micro Bypass	Canal based : Schlemm's canal	Heparin-coated non-ferromagnetic titanium stent	-
CyPass micro-stent	Suprachoroidal	Polyamide tube	Elevated episcleral venous pressure.
XEN gel stent	Subconjunctival	Gelatin	• Refractory glaucoma with failed Sx treatments. • Medically unresponsive POAG.

PRIMARY ANGLE CLOSURE GLAUCOMA

- AKA Acute Congestive Glaucoma.
- m/c age : 60-70 yrs.

Mechanism :

a. Relative pupillary block :

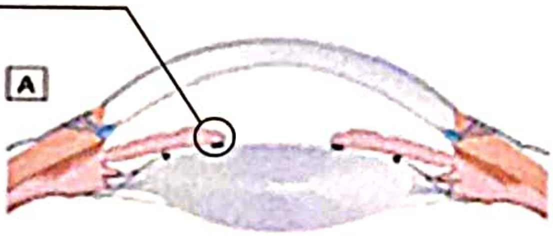
Apposition of pupillary margin against lens surface

↓

Aqueous cannot flow from PC ⟶ AC.

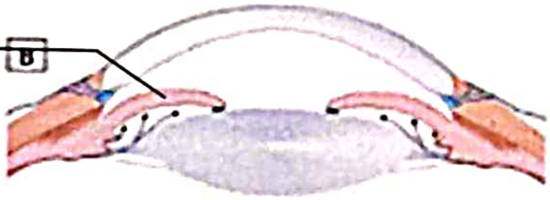
↓

Pushing effect d/t ↑ pressure in PC.



b. Iris Bombe :

Anterior bowing of iris.



c. Irido Trabecular Contact (ITC)

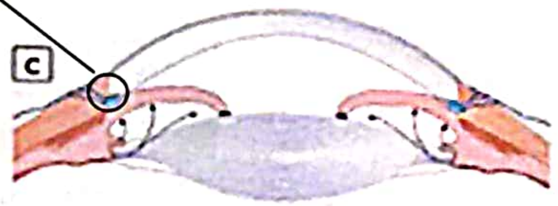
↓ If persistent

d. Peripheral anterior synechiae (PAS)

Iris adhesion with cornea and trabecular meshwork

↓

Disruption of inflow from PC to AC.



Risk factors :

D/t ↑ chance of iris opposing cornea :

- Shallow AC.
- Hypermetropia : D/t small AC.
- Females : Smaller AC.
- Plateau iris.
- Small cornea : ↓ radius with ↑ curvature.

Precipitated by dim light.

Eg : movie theatre

↓

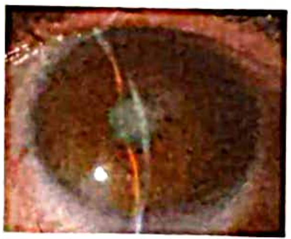
Dilation of pupil.

↓

Iris move towards angle.

↓

Angle closure.



Convexity of slit d/t iris convexity.

Classification of ACG :

	IOP	OD	VF	ITC	Rx
PACS	Normal	Normal	Normal	≥3 quadrants	Prophylactic
PAC	Raised	Normal	Normal	• ≥3 quadrants • PAS seen	Definitive
PACG	Raised	Abnormal	Abnormal	• ≥3 quadrants • PAS seen	

PACS : 1° angle closure suspect; PAC : 1° angle closure; PACG : 1° angle closure glaucoma

00:14:33

----- Active space -----

Manifestations Of PACG

SYMPTOMS

Present acutely.

- Diminution of vision.
- Redness.
- Severe pain.
- Headache.
- Vomiting.
- Coloured halos around lights.

SIGNS

Seen acutely.

IOP :

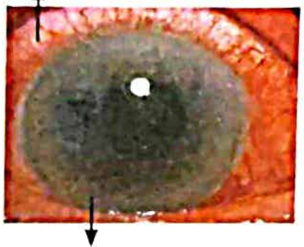
- >40 mmHg → Severe pain.
- Digital tonometry : Rock hard eye.

On torchlight :

Eclipse sign (Shallow AC).

Slit lamp findings :

1. Congestion/hyperemia :



2. Hazy cornea/Corneal edema : Breakage of tight junctions b/w endothelial cells by ↑ IOP → Corneal decompensation.

leads to

- Diminution of vision
- Coloured haloes around light d/t prismatic dispersion of light rays.

3. Vogt's triad :

Iris atrophy (D/t ↑ pressure → Ischemia)

Posterior synechiae : Adhesion b/w pupillary margin and anterior surface of lens

Glaukomflecken cataract : Anterior capsular/subcapsular opacities

↓

D/t epithelial ischemia and necrosis (2° to ↑ IOP).

4. Pupillary findings in POAG :

----- Active space -----

Vertically oval : D/t iris atrophy (Preferentially superior and inferior atrophy)

mid dilated : D/t atrophy of sphincter pupillae.

Fixed :
- Neither constricting nor dilating.
- D/t posterior synechiae effects.

5. Visual field changes :

Acute → Superior field constriction.

Chronic → Central nasal step.

6. Optic disc changes :
- Same as in POAG.
- Not visualized due to anterior chamber changes (Hazy cornea).

Treatment

00:29:39

Treated as ocular emergency.

ACUTE mx

Step 1 : ↓ IOP

a. IV mannitol (2.5%) : 1.2 g/min over 30 min.
  - DOC for acute episode/exacerbations.
  - Cannot be given long term d/t diuretic effect.

Or

b. Tab Acetazolamide 250 mg → 2 tablets stat.

Or

c. Oral Glycerin 1 g/kg of body weight.

Step 2 : miosis

DOC : Pilocarpine (Constricts pupil)
- Ineffective before IOP normalises (D/t inability to constrict if ↑ IOP).
- Ocular side effects :
  - Transient myopia.
  - Punctal occlusion.
  - Uveitis.
- Cannot be given long term d/t miosis : Impaired dark adaptation.

Note : Timolol → Nasolacrimal duct occlusion.

Step 3 : Assess corneal clarity

Clear (most cases) : Plan for SX.

Hazy (Rare) : Repeat step 1 and 2.

----- Active space -----

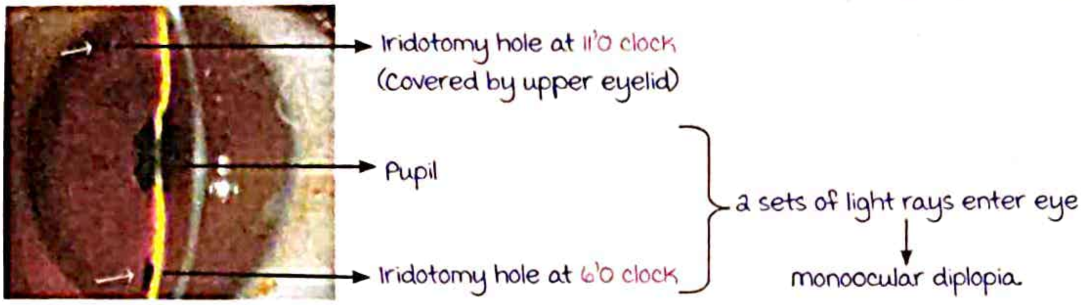
DEFINITIVE MX

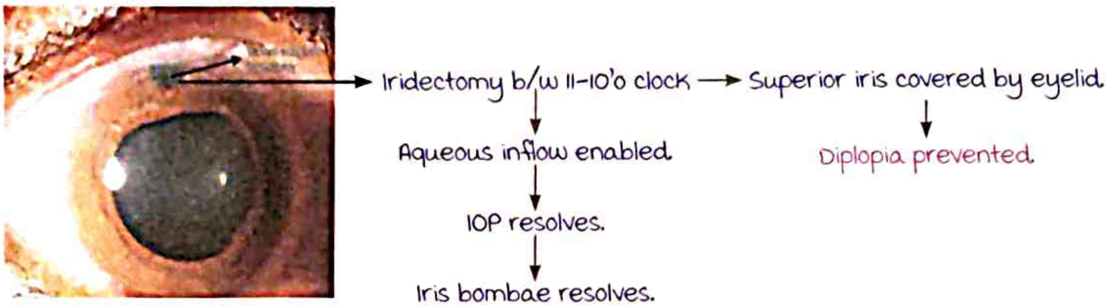
Laser iridotomy :

- Preferred over open iridectomy.
- TOC for PACG & prophylaxis of PACG.

Procedure :

- Photodisruption with Nd:Yag laser : 1064 nm.
- Creation of hole in iris b/w 11 o'clock to 1 o'clock.


Iridotomy hole at 11'O clock
(Covered by upper eyelid)
Pupil
Iridotomy hole at 6'O clock
2 sets of light rays enter eye
↓
Monoocular diplopia


Iridectomy b/w 11-10'O clock → Superior iris covered by eyelid
↓
Diplopia prevented
Aqueous inflow enabled
↓
IOP resolves.
↓
Iris bombae resolves.

Iridectomy

Rx of absolute glaucoma :

- No vision (Blind).
- Severe pain :

↓

Diode laser (810 nm) cyclophotocoagulation.

↓

Enables pain relief.

Post Sx management :

If mild ↑ IOP present (21-22 mmHg)

↓

Latanoprost.

SECONDARY GLAUCOMA(S) AND CONGENITAL GLAUCOMA

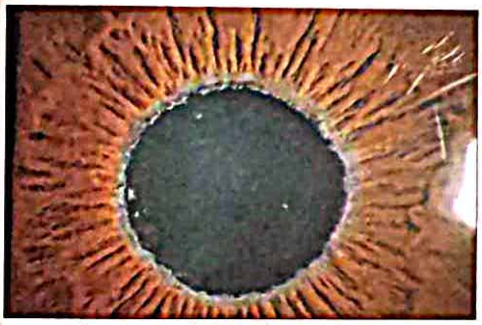
Secondary Glaucomas

00:00:12

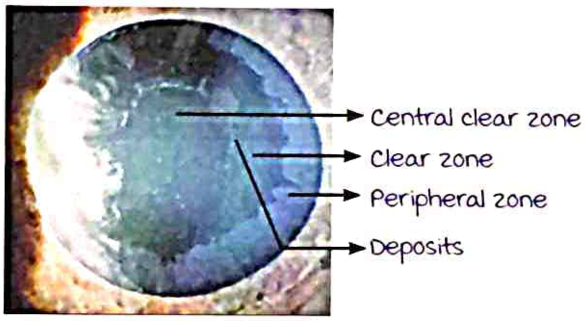
PSEUDOEXFOLIATION GLAUCOMA

Pathology : LOX-1 gene mutation → Pseudoexfoliative material produced → Deposited in different locations.

Location of pseudoexfoliative deposits	Features/Name
Trabecular meshwork	• m/c cause of 2° OAG • mostly unilateral
Pupillary margin	Fnocks : • Prevents pupillary dilatation (Poor mydriasis) ↓ Can cause difficulty during cataract Sx (Ruled out pre operatively)
Lens capsule	Target sign
Near Schwalbe's line	Sampaolesi line
Corneal endothelium	-



Fnocks : Whitish deposits over pupillary margin



Target sign

PIGMENTARY GLAUCOMA

m/c in young male myopes.

Location of pigmentary deposit	Clinical features
Corneal endothelium	Krukenberg spindle
Trabecular meshwork	2° OAG
Ciliary zonules : Insertion on lens	Zentmayer's line
Schwalbe's line	Sampaolesi line

----- Active space -----

LENS INDUCED GLAUCOMA

Types :

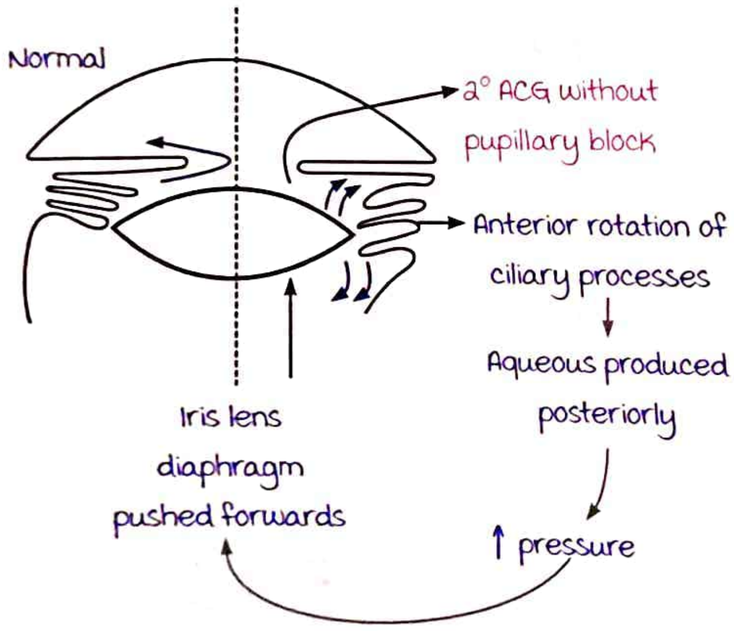
	Etiopathogenesis	Treatment
Phacomorphic glaucoma	Intumescent cataract : Swelling of lens → Contact with pupillary margin → Blocks aqueous flow → 2° ACG.	Cataract surgery
Phacolytic glaucoma	Morgagnian cataract : Liquefaction of lens proteins → Fluid leak → Blocks trabecular meshwork → 2° OAG.	
Phacotopic glaucoma	Microspherophakia : • Congenital anomaly • Small spherical lens → Laxing of zonules → ↑ lens movement → Anterior displacement : Blocks pupil → 2° ACG.	• Atropine : – Dilates pupil → Opens block • Peripheral laser iridotomy.

MALIGNANT GLAUCOMA

AKA Ciliary Block Glaucoma/Aqueous misdirection Syndrome/Inverse Glaucoma

Etiology : Incisional or laser surgeries.

Pathogenesis :


Normal
2° ACG without pupillary block
Anterior rotation of ciliary processes
Aqueous produced posteriorly
↑ pressure
Iris lens diaphragm pushed forwards

Rx :

Cycloplegic (Eg : Atropine)
↓
Relaxes ciliary muscles
↓
Zonules stretched
↓
Lens pulled posteriorly
↓
Normal aqueous flow.

Note :

Phacotopic glaucoma & malignant glaucoma : Inverse glaucoma.
(ACG Rx by atropine instead of pilocarpine)

IRIDO-CORNEAL ENDOTHELIAL (ICE) SYNDROME

1) Primary abnormality : Corneal endothelium. Silver Hammer appearance of cornea.
2) Associated finding : 2° ACG.

3 variants :

1) Progressive iris atrophy :
   - Polycoria (multiple pupils).
   - Corectopia (Abnormal pupil position).
2) Chandler syndrome : Corneal edema.
3) Cogan-Reese syndrome : Iris nevus (Pigmented lesion on iris).

Primary Congenital Glaucoma

00:23:37

Overview :

- No other associated disease.
- Age of onset : m/c <3 yrs (95%).

mechanism : Trabeculodysgenesis $\xrightarrow{\text{Causes}}$ OAG.
(TM malformation)

PRESENTATION

Symptoms :

Blepharospasm
Triad
Photophobia : Earliest symptom
Lacrimation : m/c symptom

Signs :

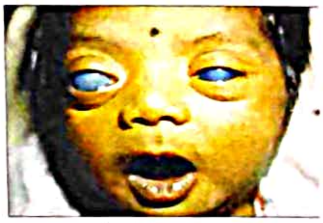
- Children's eyeball → Easily distend under pressure.
- ↑ IOP

Enlarged eyeball
↓
Buphthalmos : Enlarged eyeball
- Corneal diameter >13 mm.
(Normal 10-11 mm in children)
- Scleral stretching
↓
Thinning of sclera
↓
Underlying vessel visible
↓
Blue sclera
↓
Deep AC → ↑ Axial length → Axial myopia.

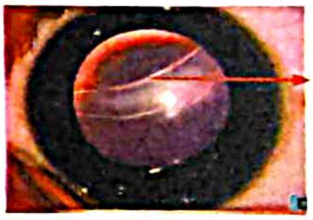
Corneal stretching

Endothelial breaks
↓
Aqueous enters cornea
↓
Hazy cornea

Horizontal breaks in Descement's membrane
↓
Haab's striae



Buphthalmos



Visible against fundal glow

Haab's striae

----- Active space -----

Gonioscopic findings :

- Anterior iris insertion.
- Barkan membrane.
- Loch Ness monster phenomenon (Loops of vessels visible).

TREATMENT

1. Rx of choice : Combined procedure.
  - Trabeculectomy (Cutting of some TM).
  - Trabeculotomy (Formation of holes in the remaining TM).
2. Goniotomy : Needle piercing of TM using a gonioscope to visualize.
  - Can be performed if cornea is clear (If onset >2 yrs).

Secondary Congenital/Developmental Glaucoma

00:35:54

Mechanism : Irido trabeculo corneal dysgenesis.

Causes :

- Axenfeld - Rieger syndrome
  - Glaucoma in 50% cases.
  - Posterior embryotoxon : Anteriorly displaced Schwalbe's line.
- Peter's anomaly.
- Aniridia : Ectopia lentis and glaucoma.
- Phacomatosis : NF, Sturge Weber syndrome.

TESTS FOR VISION AND NORMAL OPTICS OF EYES

Components of visual acuity/Vision

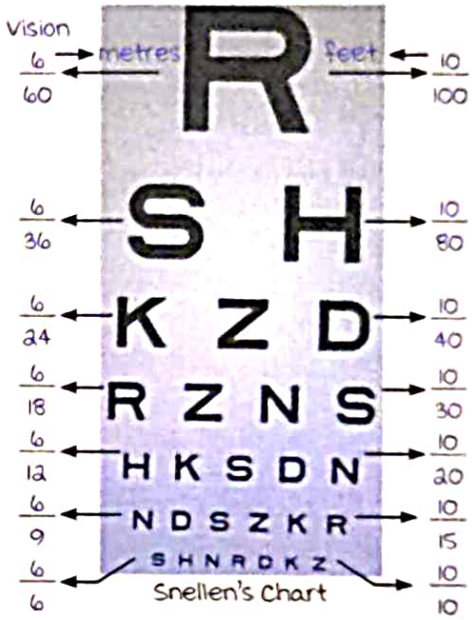
- Minimum visible
- Minimum resolvable
  - Tested clinically
  - MAR (minimum angle of resolution) : 2 points subtending an angle of
    - < 30-60 seconds — On eye : Can't be viewed separately
    - >30-60 seconds — On eye : Can be viewed separately
- Minimum discriminable / hyperacuity
  - Cannot be measured by clinical tests
  - Eg : - stereopsis (3D vision)
    - vernier acuity

Tests for Vision

00:07:40

TESTS FOR FAR VISION

I. Snellen's chart :



Snellen's Chart

- Minimum 7 rows of letters.
- Number of letters per row : Variable.
- Each eye tested separately.

Fraction at the rows :

eg :- $\frac{6}{24}$ → 6 → Distance of examination (in m).

24 → Respective distance: minimum distance at which normal eye can visualize the row.

Procedure :

Patient positioned at 6m/20ft. from chart

↓

Serial attempts to read all rows until smallest visible row $\xrightarrow{\text{If visible}}$ Record acuity - eg : 6/9

↓ If even 6/60 is not visible at 6m

Move patient closer in increments of 1m until larger row is visible $\xrightarrow{\text{If visible}}$ Record acuity -eg : 3/60 if largest row seen at 3m

↓ If not visible until 1m from the chart

Finger counting close to face $\xrightarrow{\text{If visible}}$ FCCF +

↓ If negative

Perception of hand movements tested $\xrightarrow{\text{If visible}}$ HM (+)

↓ If not perceived

Perception of light tested $\xrightarrow{\text{If visible}}$ PL +

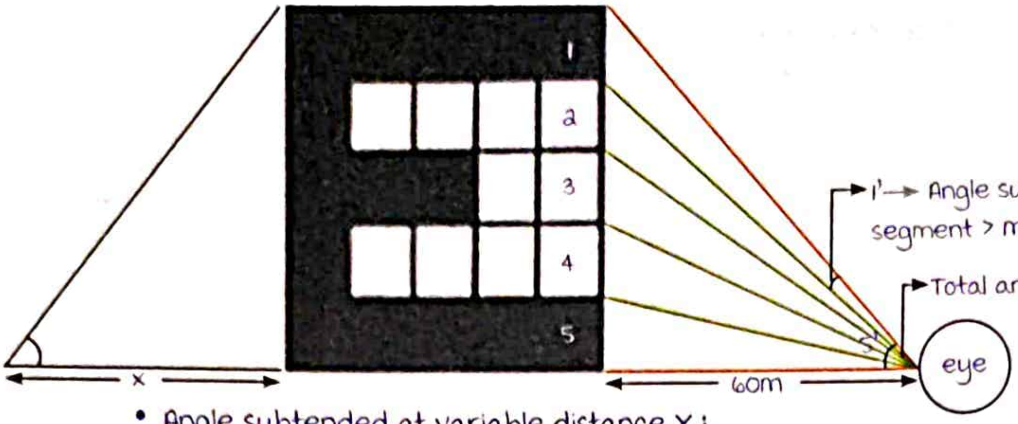
↓ If negative

Patient blind

----- Active space -----

Note : Least acuity discernible via Snellen's chart : 1/60.

Principle : Each optotype subtends angle of 5 minutes of arc at nodal point of eye when viewed from its respective distance.


1
2
3
4
5
1' → Angle subtended by each segment > MAR
Total angle → 5'
5'
x
60m
eye

- Angle subtended at variable distance x :

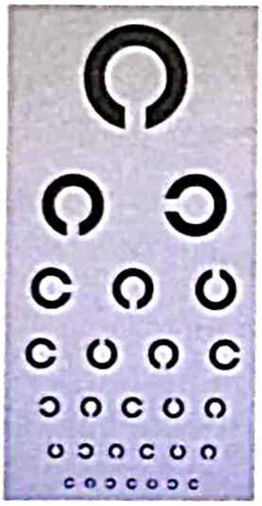
$$\text{Angle (in minutes)} = \frac{\text{Respective distance}}{\text{Given distance }(x)} \times 5'$$

2. Direction identification tests :

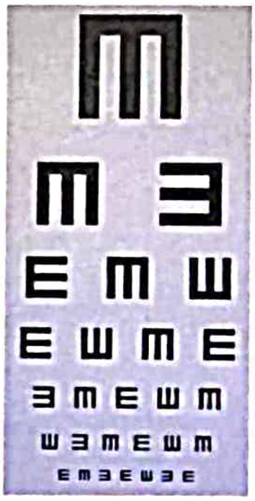
Used in illiterate patients.

Types :

a. Landolt C ring chart



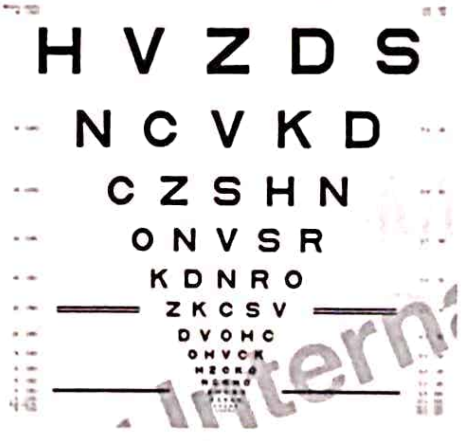
b. Snellen's E chart



3. Bailey-Lovie / logMAR chart :

- Number of letters per row: constant (5)
- Log value used to record acuity
- Not used in India

Advantages : - No crowding phenomenon
- Increased accuracy at lower visual acuity


H V Z D S
N C V K D
C Z S H N
O N V S R
K D N R O
Z K C S V
D V O H C
O H V C K

Bailey love chart

4. Tests for verbal fx - school children (age 2-5 yrs) :

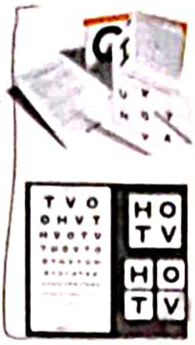
Do not require communication by patient.

Types

Based on matching of letters :

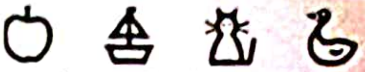
a. Sheridan Gardiner : HOTV test
   - Child matches letters on the cards/tiles →

b. STYCAR : Screening in young children & the intellectually disabled

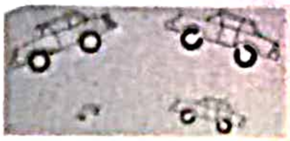


Based on matching of pictures :

a. Key Picture test :



b. Broken wheel test



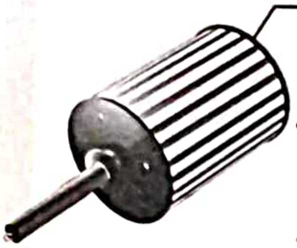
c. Allen Picture card pre school test

5. Tests for infants

a. Optokinetic Nystagmus (OKN) tests :

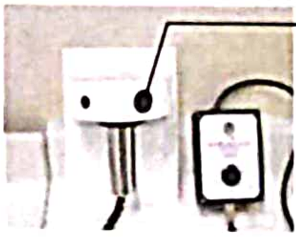
- Done with rotating drum → Infant fixates gaze on dot/strip as it appears & disappears.

OKN Drum :



- Thinner the strip perceived
  ↓
  Higher the acuity
- Resolvable acuity (MAR) tested

Carford drum :



- Smaller the dots visualized
  ↓
  Higher acuity
- Detection acuity tested

b. Preferential Looking Test (PLT) :

- An infant prefers to look at striped over plain areas.
- Progressive narrowing of stripes.
- Thinner the stripes visualized → ↑ acuity.

Types of PLT

Teller acuity cards

Keeler gratings

c. Cardiff Acuity Test (CAT) :

Used in both infants & toddlers.

Based on vanishing optotype.

CAT

D. Visual evoked potential (VEP) :

Principle: Light stimulus → Visual pathway → ↑ Visual cortex activity → Normal VEP.

----- Active space -----

e. Indirect assessment :

Reflex	Components	Time of development
Blink reflex	- Stimulus : Illumination of eyes - Response : Blinking of eye	Present since birth
menace reflex	Stimulus : Approach of an object near eye Response : Closure of eyes	Develops around 5 months of age

TESTS FOR NEAR VISION

Use : for diagnosis of presbyopia

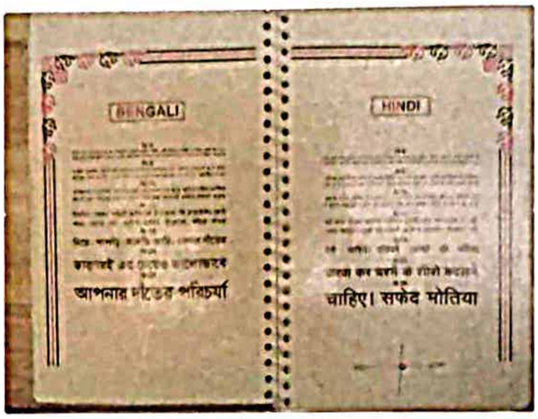
Distance of examination: 30-35 cm (comfortable reading distance).

a. Snellen's near chart (m/c) : N_6 (best), N_9, N_{12}, N_{24}, N_{36} (worst).

b. Jaeger's chart : Results between $J_1 \rightarrow J_7$.

c. Roman test type

Note : least distance of distinct vision (Near point) = 25cm.



Snellen's near chart

TESTS FOR CONTRAST SENSITIVITY

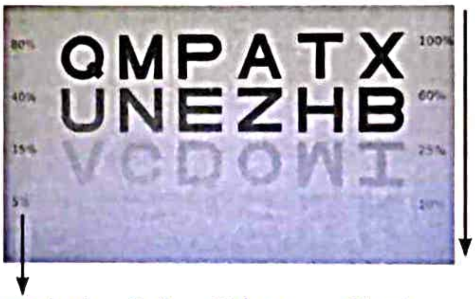
Use : Diseases like cataract

↓

Contrast sensitivity affected before visual acuity

Tests : mnemonic → CRAP BF

- Cambridge low contrast gratings
- Regan chart
- Arden gratings
- Pelli-Robson chart (m/c) →
  - Letters as triplets
  - Distance of examination : 1m
- Bailey-Lovie contrast chart
- FACT chart.



Reducing contrast

% correctest Pelli Robson Chart

Normal Optics of Eye

00:58:42

Function of eye : converging of light rays onto retina.

Areas with refractive power :

a. Gullstrand's Concept :

	Cornea		Lens	
	Anterior surface	Radius of curvature	Anterior surface	Posterior surface
Refractive powers	+48.8 D	-5.88 D	+19 D	
Radius of curvature	7.7 mm	6.8 mm	10 mm	-6 mm

Note : Power at posterior surfaces of cornea & lens are in the negative as light is moving from denser to rarer medium.

b. Listing's reduced eye :

- Total power of :
  - Eye : +58.6 D
  - Cornea : +43 D
  - Lens : +19 D
- Application :

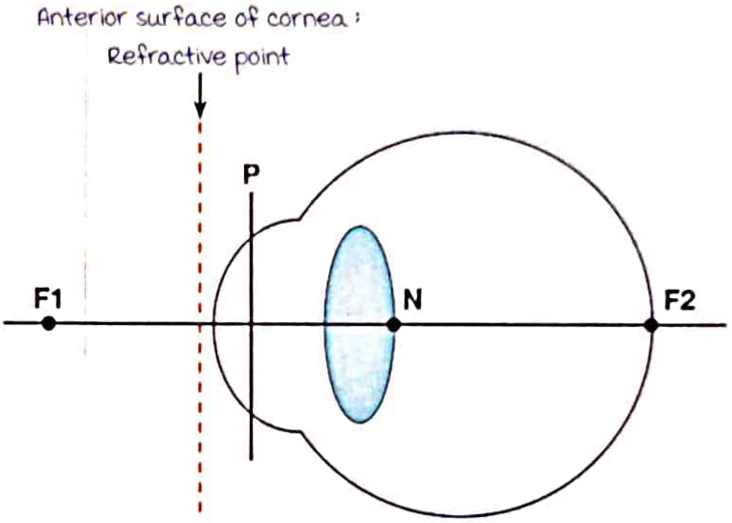
  Alter axial length

  ↓

  N, P, F alteration

  ↓

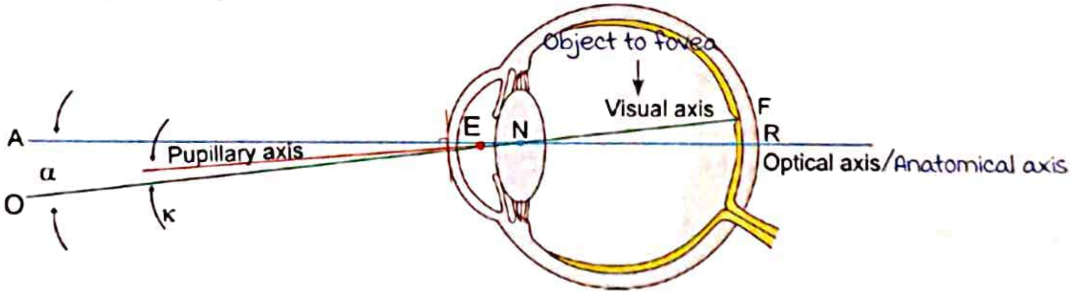
  Aberrant image formation
  (not on retina)


Anterior surface of cornea :
Refractive point
P
F1
N
F2

P : Principle point (1.35mm from reference)
N : nodal point (7.0mm from reference)
F_a : Focal point for real image (24.13mm from reference)
F_i : Focal point for virtual image (-15.7mm)

Factors affecting refracting Power :

- Radius of curvature.
- Difference in refractive index b/w 2 media.
- Axial length of eye.


Object to fovea
Visual axis
A
Pupillary axis
α
O
K
E
N
F
R
Optical axis/Anatomical axis

a. Angle Kappa (K) : B/w visual & pupillary axis.
- Normally ≤5° (if ↑↑ or ↓↓ → appearance of deviated eyeball.

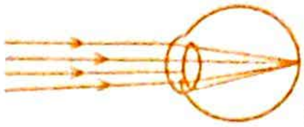
b. Angle alpha (α) : B/w visual axis & anatomical axis.

MYOPIA AND HYPERMETROPIA

Emmetropia :

Normal refractory power of eye (+58.6 D).

Parallel rays from infinity



Converge on retina

Ametropia :

Abnormal refractory power.

Mono-ocular

Present in monocular and binocular vision.

Spherical

Corrected by spherical lens.

- Myopia
- Hypermetropia

Cylindrical

Corrected by cylindrical lens.

Astigmatism.

Binocular

- Pathology contributed to by both eyes.
- Present only in binocular vision.
- Disappears in monocular vision.
- Eg:
  - Anisokonia
  - Anisometropia

Pathogenesis and Mechanism of Spherical Refractory Errors 00:06:30

Myopia :

Pathogenesis :

↑Refractory power of eye
↓
Early convergence of rays.
↓
Image formed anterior to retina.

Corrective lenses : (–) lenses.

Hypermetropia :

Pathogenesis :

↓Refractory power of eye
↓
↓ converging of rays.
↓
Image formed behind retina.

Corrective lenses : (+) lenses.

mechanisms of change in refractive power of eye :

	myopia	Hypermetropia
a) Axial length (AL) (Normally 24mm)	Axial myopia (m/c) : • ↑Axial length. • 1mm ↑in AL = 3D added to power. • Eg : Buphthalmos.	Axial hypermetropia : • ↓Axial length. • 1mm ↓ in AL = 3d subtracted from power.
b) Curvature of cornea/lens (Radius of curvature : ROC)	Curvatural myopia : • ↑Curvature of cornea/ lens : ↓ROC. • Eg : Keratoconus/Lenticonus.	Curvatural hypermetropia : ↓curvature of cornea/lens : ↑ROC.
c) Refractive index (RI) of lens>cornea.	Index myopia : ↑RI of lens. Eg: Nuclear cataract (sclerosis).	Index hypermetropia : ↓RI
d)Position of lens (Power =1/ focal length)	Positional myopia : • Lens positoned anteriorly. • Focal length : ↓ • Power of lens : ↑ • Eg: Weill marchesani syndrome → Lens moves forwards → ↓focal length & ↑power	Positional hypermetropia : • Lens positioned posteriorly. • Focal length : ↑ • Power of lens : ↓

Presentations of Spherical Refractory Errors

00:20:09

SYMPTOMS

In myopia :

- Loss of far vision : Near/short-sightedness.
- Floaters present in high myopia : D/t vitreous syneresis/liquefaction.
- Half closure/Half squinting of eyes
  - Cutting off of peripheral rays → Central rays fall straight onto retina → Improves vision

In hypermetropia :

Loss of near vision (far sightedness) : Clinically no difficulty in near vision d/t over-accomodation → Presents as tired eyes/headache : Asthenopia.

EXAMINATION SIGNS

	Myopia	Hypermetropia
Size of eyeball	Large (↑AL) → Pseudoproptosis	Small (↓AL)
Anterior chamber	Deeper than normal	Shallower than normal : Prone to angle closure glaucoma.
Squint	Far point comes near to eye ↓ Accomodation not required for near vision (Convergence does not occur after) ↓ Convergence insufficiency ↓ Propensity to deviate outwards : Exophoria.	Overaccomodation ↓ Overconvergence ↓ Esotropia
Angle Kappa	Lesser than normal → Pseudoesotropia : Appearance of inward deviation of eye	more than normal (>5°) → Pseudoexotropia : Appearance of outward deviation of eye.

FUNDOSCOPY SIGNS

Fundus : View of all structures visible together in posterior segment of eye.

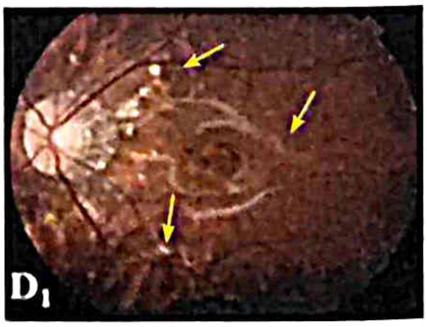
- Vitreous. } Transparent
- Retina } Transparent
- Choroid : Red

Hypermetropia :

- Shot silk fundus : Shiny appearance.
- Pseudo papillitis.

Myopia :

- Signs seen if pathological/degenerative myopia present :
  - AL>26mm OR
  - Refractive power >+6D.
  - a. Tessellated/Tigroid appearance :
    - Diffuse attenuation of retinal pigment epithelium.
    - Choroidal red color more visible.



Tessellated/Tigroid appearance

b. Temporal myopic crescent :

Disparity in stretching of eyeball
↓
Chorioretinal atrophy
↓
Underlying sclera visible (White)

c. Lacquer cracks :

Fine breaks in Bruch's membrane
(Innermost layer of choroid)
↓
Underlying sclera visible (Yellowish)

d. Foster Fuch spots : Areas of subretinal hemorrhage at macula.

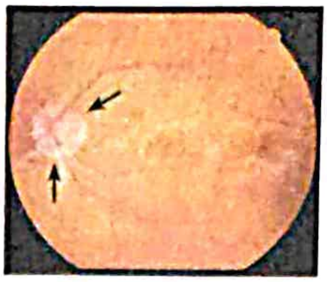
e. Posterior staphyloma : In sclera.

↑AL → Scleral stretching → Scleral thinning → Bulging d/t normal IOP → Staphyloma (Ectatic bulge in sclera).

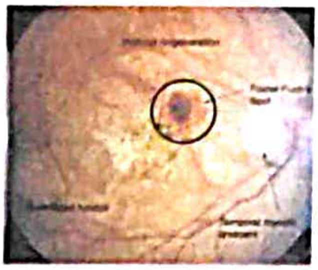
f. Rhegmatogenous retinal detachment :

- m/c complication of pathological myopia.
- ↑AL → retinal stretching.
  - Break in retina (detachment)
  - Rupture of vessels : Vitreous hemorrhage.
- Supertraction : Vitreous pulls retina over optic disc (OD) → OD covered by retina → Tilted OD (seen in very high myopia).

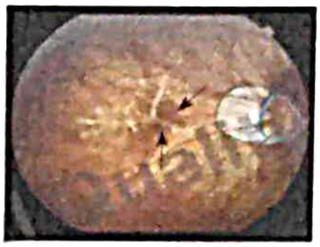
----- Active space -----



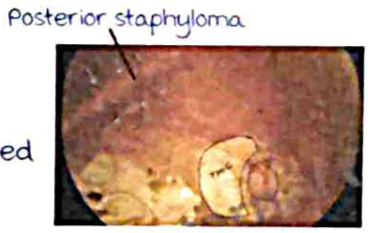
Temporal myopic crescent



Foster Fuch's spots

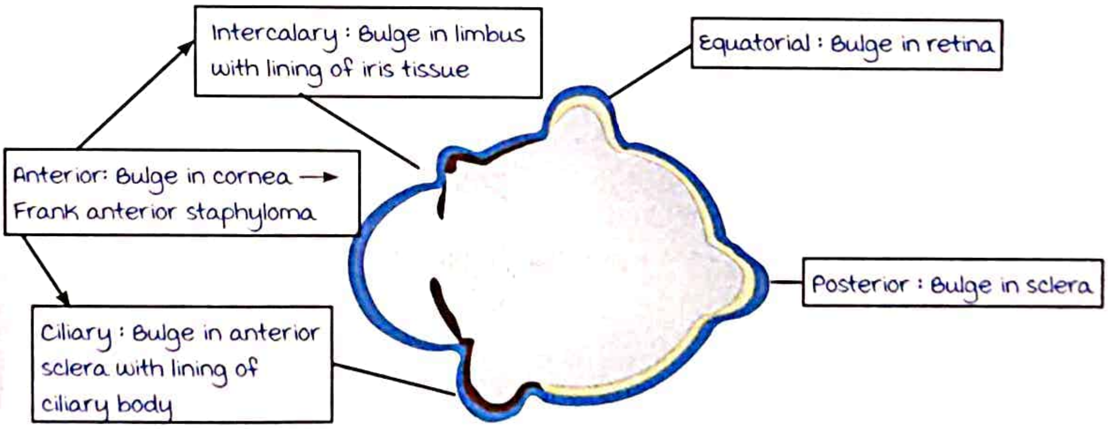


Lacquer cracks


Posterior staphyloma

myopic fundus

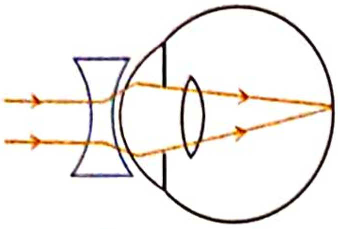
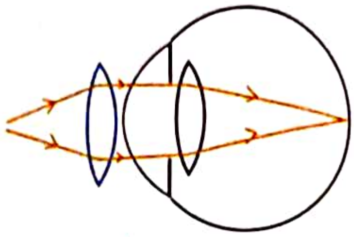
Note : Types of staphyloma


Intercalary : Bulge in limbus with lining of iris tissue
Equatorial : Bulge in retina
Anterior: Bulge in cornea → Frank anterior staphyloma
Posterior : Bulge in sclera
Ciliary : Bulge in anterior sclera with lining of ciliary body

----- Active space -----

Spectacles & Lenses

00:57:30

Treatment	Myopia	Hypermetropia
Spectacles : Spherical	 Concave/diverging glasses (with negative power)	 Convex/converging lens (with positive power)
Contact lenses	Soft lenses • Preferred in high myopia because of high powered concave glasses. – Are thicker. – minify image.	-

Surgery for Hypermetropia

01:00:08

Rarely performed d/t :

- Incidence of hypermetropia < myopia.
- Very high hypermetropia is rare.

1. Thermal laser keratoplasty : THC : YAG laser.
   (THC : Thallium Holmium Chromium)
2. Hyperopic LASIK.
3. Conductive keratoplasty : using radiofrequency

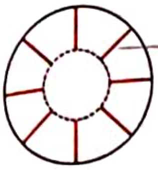
Surgery for Myopia

01:03:11

RADIAL KERATOTOMY

Principle : ↓corneal curvature ⟶ ↓refractive power.

Procedure :



Cornea

8 partial thickness peripheral corneal incisions

↓

Healing by fibrosis

↓

Contraction causing stretching of cornea

↓

- ↓ thickness of cornea.
- ↓ of refractive power.

- Not done currently d/t unpredictable results.

LASER SURGERY

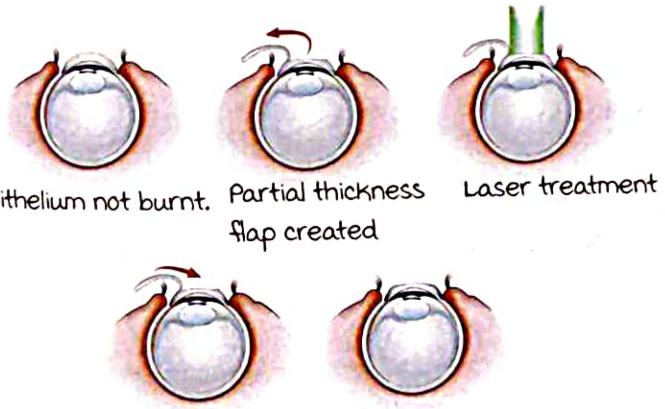
1. **PRK : Photo Refractive Keratectomy.**
2. **LASIK : Laser Assisted In-Situ Keratomileusis**

} Excimer laser used

Excimer laser :

- Argon flouride laser.
- Wavelength : 193nm.
- MOA : Photablation ⟶ Central thinning of cornea ⟶ Flattening of cornea ⟶ ↓Refractive power.

LASIK procedure :



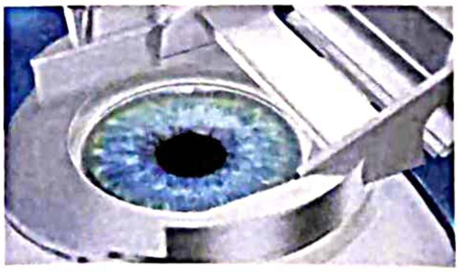
Epithelium not burnt.

Partial thickness flap created

Laser treatment

Reattach flap

microkeratome



Creates epithelial flap

- 10-12 microns ablated for each dioptre.
- 250 micron stromal bed residual thickness.

C/I to LASIK :

- Corneal thickness <450µm pre-operation : ↑chance of breaching residual thickness barrier.
- Age < 18yrs.
- Refraction is not stable for at least 12 months pre-op.

3. SMILE : Small Incision Lenticule Extraction :

- Laser used : Nd : Glass
- mechanism : Photodisruption.
- Flapless surgery ⟶ No side effects from flap creation.

Procedure of SMILE :

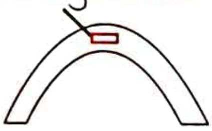
1. Patient positioned near machine.
2. machine creates suction around cornea : Ensures eye does not move.
3. Laser fixed:

Eye visualised

Stabilisation by suction

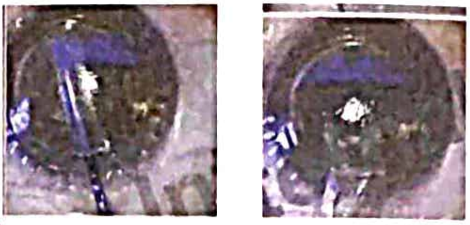
2 incisions given near stroma :

- Deep.
- Superficial.



↓

Free scleral stromal button created : Lenticule.



Pulling out of the lenticule

4. Corneal Incision: Lenticule pulled out ⟶ Thinned cornea.

ICL SURGERY

Implantable collamer lens (ICL)

- made of hydrogel.
- Implanted in the sulcus anterior to lens.

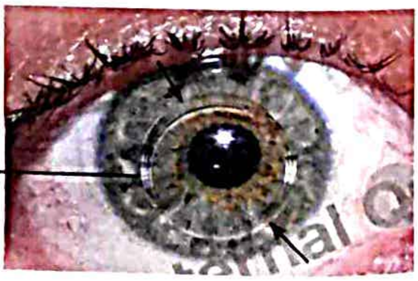
Indication : myopia >8D exclusively (high myopia).

- LASIK in high myopia ⟶ ↑chance of breaching stromal residual bed thickness criteria.

INTACS

Intra Corneal Stromal Ring Segments

- Can correct 2-5D pf myopia (low power myopia)



2 semi-circular rings implanted ⟶ Stretches & flattens cornea.

Note : Convex v/s concave lens.

----- Active space -----

	Convex lens	Concave lens
Refraction	• Converging lens. • (+) lens.	• Diverging lens. • (–) lens.
Identification	• High power lenses: - Thick in centre. - Object appears maginified. • On moving convex lens : Object moves in opposite direction.	• High power lens : - Thick at periphery. - Object appears minified. • On moving concave lens : Object moves in same direction.
uses	• Correction of hypermetropia, aphakia, presbyopia. • Ophthalmoscopy (+20D in indirect).	Correction of myopia.
Image	• Real and inverted (m/c). • may produce virtual and arect image.	Always virtual, erect, diminished image.

----- Active space -----

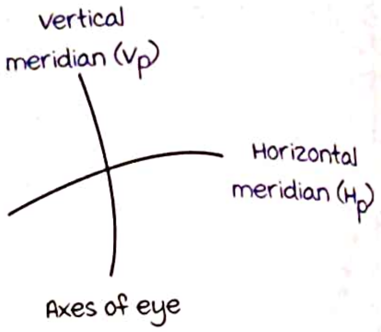
ASTIGMATISM, READING OF SPECTACLE PRESCRIPTION AND BINOCULAR ERRORS

00:00:13

Astigmatism (A)

Overview :

- Cylindrical refractory error.
- Different refractory power in both principal meridians/axes of eye.
  (In spherical refractory errors → Same power in both axes).



CLASSIFICATION

- Regular astigmatism : Both principal meridians are perpendicular to each other.
  - With the rule A : Vertical meridian more curved.
  - Against the rule A : Horizontal meridian more curved.
  - Oblique A : Principal meridians perpendicular to each other, but not at 90° and 180°.
- Irregular astigmatism :
  - Both principal meridians are not perpendicular to each other.
  - Can be due to pathologies : Eg : Corneal scar, cataract, etc.

REFRACTIVE TYPES

	Axis 1	Axis 2
Simple myopic	Emmetropic	Myopic
Compound myopic	Myopic	
Simple hypermetropic	Emmetropic	Hypermetropic
Compound hypermetropic	Hypermetropic	
Mixed astigmatism	Myopic	Hypermetropic

CLINICAL FEATURES

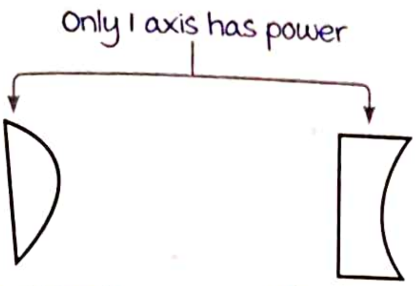
1. Loss of vision : Loss of sharpness (All letters seem to stretch/merge).
2. Asthenopia

TREATMENT

1. Spectacles :

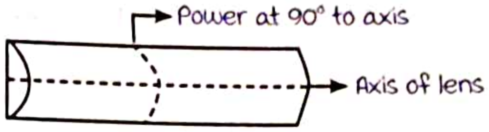
Cylindrical lenses.

- Have power 90° to their axis.
- Types :


Only I axis has power

Plano convex : For hypermetropic A

Plano concave : For myopic A

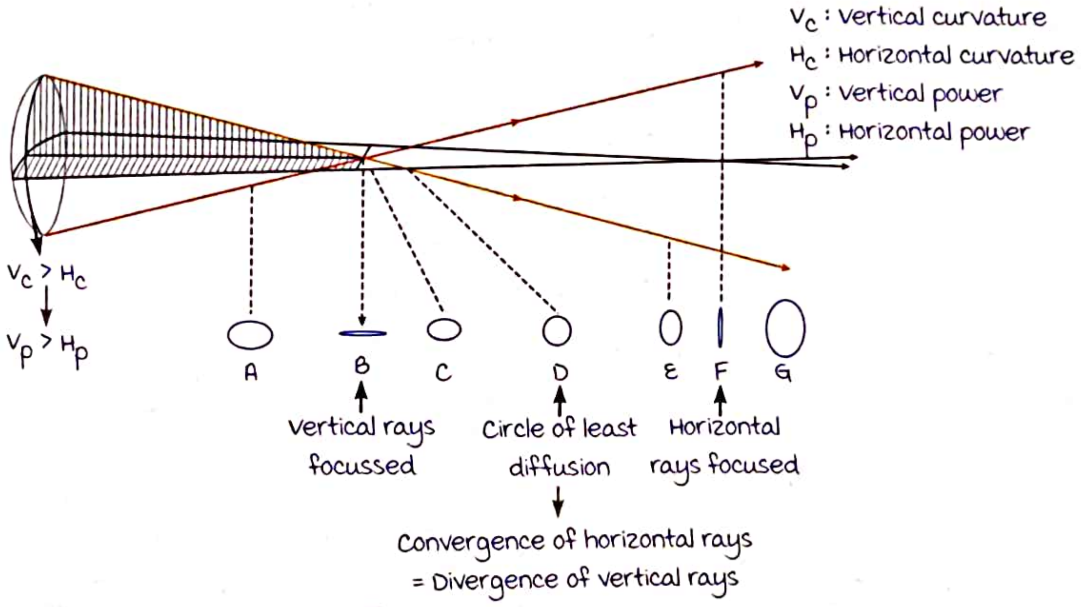

Power at 90° to axis
Axis of lens

2. Toric Contact Lenses :

A lens with 2 surfaces with different powers.

Sturm's Conoid :

Arrangement of light rays refracted through a toric surface.


V_c : Vertical curvature
H_c : Horizontal curvature
V_p : Vertical power
H_p : Horizontal power
$V_c > H_c$
$V_p > H_p$
A
B
C
D
E
F
G
Vertical rays focussed
Circle of least diffusion
Horizontal rays focused
Convergence of horizontal rays = Divergence of vertical rays

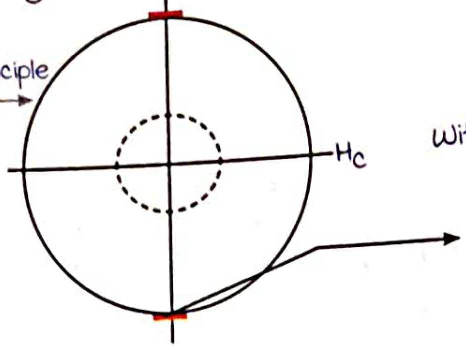
Principle : Converges rays so circle of last diffusion lies at retina ⟶ Clear image visualized.

----- Active space -----

3. Surgery :

a. Astigmatism Lasik : Selective burning off of the axis with higher curvature in the cornea.

b. Accurate Keratotomy
c. LRI (Limbal relaxing incisions)
} Principle



Eg :

With the rule astigmatism ($V_c > H_c$)

↓

Partial thickness incision at 90° & 180° on cornea/limbus

↓

Heals by fibrosis

↓

Vertical curvature stretched

↓

Reduced V_c

Deciphering Spectacle Prescriptions

00:21:17

Format of prescription :

	Sph	Cyl	Axis
RE			
LE			

- RE : Right eye.
- LE : Left eye.
- Sph : Spherical power $\xrightarrow{For}$ myopia/hypermetropia
- Cyl : Cylindrical power $\xrightarrow{For}$ Astigmatism.
- (+) power prescribed for hypermetropia
- (–) power prescribed for myopia

Spherical and cylindrical lenses :

	Lens type	Example of prescription
myopia	– sph	–1D sph
Simple myopic astigmatism	– cyl	–1D cyl
Compund myopic astigmatism	– sph/– cyl	–1D sph/–1.5D cyl
Hypermetropia	+ sph	+0.75D sph
Simple hypermetropic astigmatism	+ cyl	+1.5D cyl
Compound hypermetropic astigmatism	+ sph/+ cyl	+0.5D sph/+1 D cyl
mixed astigmatism	– sph/+ cyl + sph/– cyl	–1D sph/+2D cyl +1D sph/–2D cyl

Simple transformation :

Done to ensure that cyl power > sph power

Eg :

- In a prescription −x D sph / +y D cyl @ 180° where x > y.

↓

Apply simple transformation.

  - New sph : Old sph + old cyl.
  - New cyl : −(Old cyl).
  - New axis : Perpendicular to old axis.
- New prescription : (y−x) D sph/−yD cyl @ 90°.

With and Against the rule astigmatism :

Inferred using the axis of cylindrical lens being prescribed.

	180°	90°
With the rule	− lens (c)	+ lens (v)
Against the rule	+ lens (v)	− lens (c)

C : Convex lens.

V : Concave lens.

- Eg : −1D cyl @ 180° (cylindrical lens with axis of 180°)

↓

↑ power at 90° (As power of cylindrical lens is perpendicular to its axis)

↓

Vertical power > horizontal power : With the rule astigmatism.

Binocular Refractory Errors

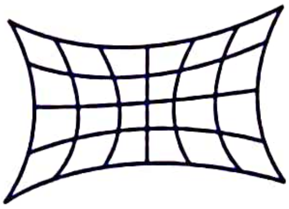
00:46:45

ANISEIKONIA

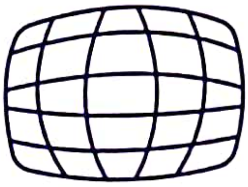
- Different shape (Distortion) and/or size of an image between both eyes.
- Occurs d/t varying thickness of glasses :
  - Convex lens : Thicker at center.
  - Concave lens : Thick at periphery.

Pin cushion defect : Seen with very high plus (+) correction.

Barrel defect : Seen with high minus (−) correction.



Pin cushion defect



Barrel defect

----- Active space -----

ANISOMETROPIA

- Different refractory power b/w both eyes.
- Significant if difference >2.5 D.

Magnification rule for convex lenses for 1D → 2% image magnification $\xrightarrow{\text{if significant anisometropia}}$ Varying sizes of images from each eye → Failure to fuse 2 images : Diplopia

Rx : Surgical correction.

REFRACTION : HOW TO PRESCRIBE GLASSES AND APHAKIA

Refraction : Procedure of determining and correction of refractive errors.

Types :

Objective refraction :

- Performed first.
- Independent of patient responses.

Subjective refraction :

- Performed second.
- Patient's response taken into account.

Objective Refraction

00:02:21

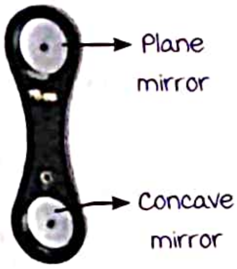
Performed in Retinoscopy/Skiascopy/Shadow test.

methods :

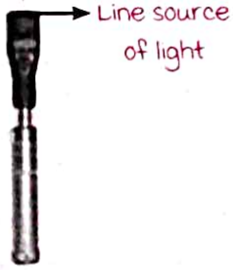
Retinoscopy methods

- Manual retinoscopy
  - Instruments employed
- Automated refractometers

Reflecting mirror retinoscope (Obsolete)



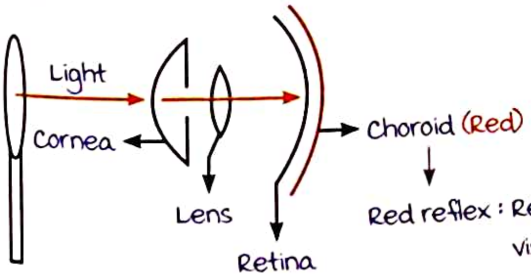
Priestley-Smith retinoscope : Contains 2 mirrors.



Self illuminated STREAK retinoscope : Battery operated.

PROCEDURE OF RETINOSCOPY

Using self illuminated STREAK retinoscope.



Red reflex : Red streaks of light visible to examiner.

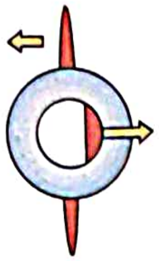
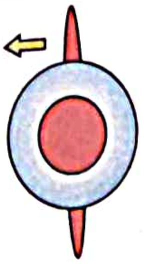
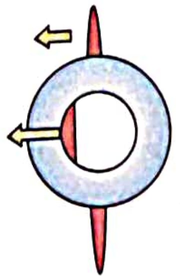
Self Illuminating Streak retinoscope

----- Active space -----

Distance of examination : 1 metre.

Method : Examiner moves retinoscope → Direction of movement of red streak compared to movement of retinoscope.

Findings :

	Against movement	No movement	With movement
Appearance			
Dx	> 1D myopia	1D myopia	< 1D myopia/Emmetropia/Hypermetropia
Refractive power scale	−3 −2 Against movement	−1 No movement	0 1 2 3 With movement

RETINOSCOPY VALUE

Determined by neutralization.

Neutralization :

Procedure : movement of red reflex seen on retinoscopy
↓
Place lens in front of eye during retinoscopy
↓
Power of lens at which movement ceases (neutralization) is noted.

Influence : Power of lens at neutralisation = RV.

- R.V. is 0 at +1D (If no lens used).

Corrected Retinoscopy Value (CRV) :

Takes into account :

1. Variation in distance of examination (d) : $C_1 = 1/d$
2. Addition of cycloplegics/mydriatics :
   - Correction for tonal allowance.
   - C_a =
     a. 1 : Atropine
     b. 0.75 : Cyclopentolate
     c. 0.50 : Homatropine

Formula : $CRV = RV - C_1 - C_a$

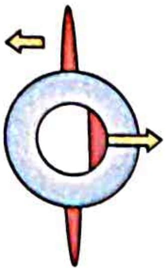
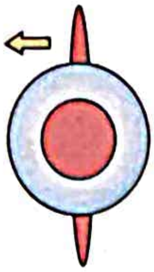
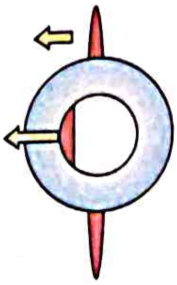
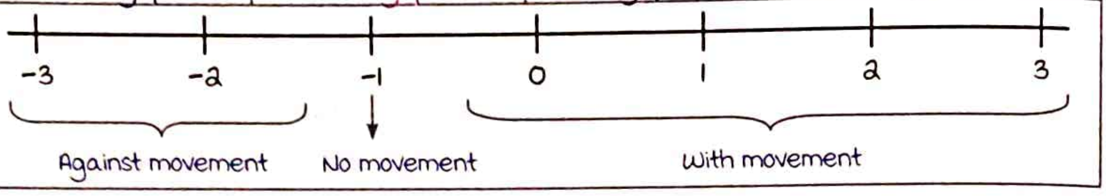
- CRV can be calculated in different axes.

----- Active space -----

Distance of examination : 1 metre.

Method : Examiner moves retinoscope → Direction of movement of red streak compared to movement of retinoscope.

Findings :

	Against movement	No movement	With movement
Appearance			
Dx	> 1D myopia	1D myopia	< 1D myopia/Emmetropia/Hypermetropia
Refractive power scale	 -3 -2 -1 0 1 2 3 Against movement No movement With movement		

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   - Correction for tonal allowance.
   - C_a =
     a. 1 : Atropine
     b. 0.75 : Cyclopentolate
     c. 0.50 : Homatropine

Formula : CRV = RV − C_1 − C_a

- CRV can be calculated in different axes.

Note : Cycloplegia always done during retinoscopy (D/t interference from accommodation).

- In children < 5 yrs : DOC ⟶ 1% Atropine eye ointment (TDS for 3 days).
- In adults :
  - DOC : 0.5% – 1% Cyclopentolate eye drops.
  - m/c used : Tropicamide (D/t quick and short acting).

Prescribing spectacles

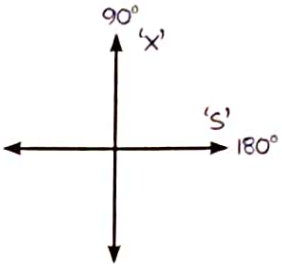
00:27:00

method :

Step 1 : Choose any axis as 'S'.

Step 2 : make other axis as 'X'.

Step 3 : Calculate for prescription.



Where X and S are CRVs in their respective axes

Format for prescription of spectacles :

- Spherical CRV/Cylindrical CRV @ axis of spherical.

S/X–S at axis of S or X/S–X at axis of X

Subjective refraction

00:37:07

Assesses if patient is comfortable with power calculated on objective refraction.

Adjustment of refraction :

- Trial and error method.
- Fogging technique.

Refinement of refraction

Cylinder :

- Jackson cross cylinder test.
- Astigmatic fan test.

Sphere :

- Duochrome test.
- Pinhole testing.

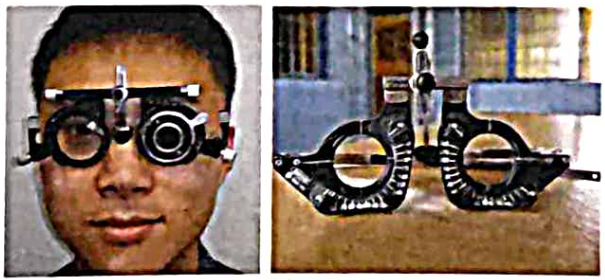
Binocular balancing :

- Duochrome test with fogging.
- Prism dissociation method.

ADJUSTMENT OF REFRACTION

1. Trial and error method :

- Primary method using a trial frame.
- Lens power altered till Snellen's chart is clearly visible.
- Uniocular testing.



Trial frame : Contains multiple slots for spherical & cylindrical lenses.

2. Fogging method :

Use :

- To overcome abnormally high accommodation.
- Before cycloplegia.

----- Active space -----

Procedure : Placing very high convex lens (+ power).

↓

Relaxes ciliary muscles (Relaxes accommodation).

↓

True refractive error can be assessed.

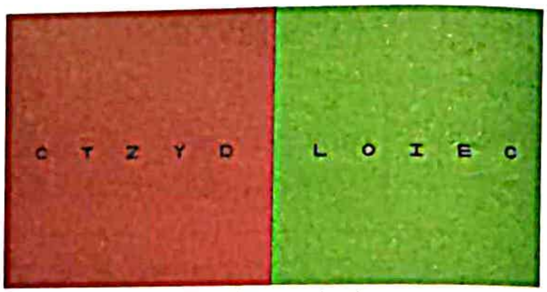
REFINEMENT OF REFRACTION

Detection of minute errors in prescription.

Refinement of spheres :

1. Duochrome test :

Appearance	Inference
Letters equally clear against both backgrounds	Correct spherical power prescribed
Letters on red background appear clearer	Residual myopia : Indicates undercorrection
Letters on green background appear clearer	Residual HMT : Indicates overcorrection



Duochrome test

2. Pinhole test :

Pinhole : 1 mm diameter

Uses	Procedure	Results & Inference
Refinement/ Confirmation of spherical power	After prescription of spectacles ↓ Pinhole kept in front of eye with spectacles.	If vision improves further ↓ Indicates residual refractory error upto 4D.
To determine presence/absence of refractory error	When patient complains of LOV ↓ Pinhole kept in front of eye.	• Vision improves : Indicates refractory error as cause of LOV. • Vision does not improve: Indicates other ocular pathology as cause of LOV.
Perform 2-point discrimination test / macular function test	Disc with 2 pinholes 2mm apart ↓ Single light source shown to patient.	Normal macula : Patient sees 2 separated light points.
Low vision aid	multiple holed pin hole used	-

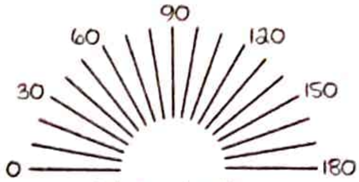
----- Active space -----

Refinement for cylinders :

1. Jackson's Cross Cylinder Test :
   - Power :
     -2.5 D Sph/+0.5 D Cyl or -0.5 D Sph/+1 D Cyl.
   - Create effect if a cross cylinders :
     - minus cylinder on 90° axis.
     - Plus cylinder on 180° axis.

Jackson's Cross Cylinder

2. Astigmatic Fan Test :
   method : view fan with spectacles.



Astigmatic Fan

All lines appear equally dark/clear
↓
Appropriate prescription provided

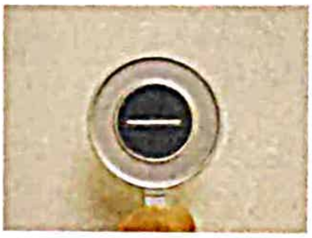
Any line looks darker/clearer
↓
Refinement needed in cylindrical prescription.

Stenopaic Slit (SS)

00:55:15

uses

1. Diagnosis of astigmatism :



Stenopaeic slit

Procedure : SS kept in front of eye ⟶ Rotated slowly for 360°.

Observation & Inference : If patient sees clear image at a particular point
↓
Indicates presence of astigmatism.

2. Confirmation of power of astigmatism :

Procedure : Cut hole in axis of cylinder while patient wears spectacles prescribed.

Observation & Inference : If vision improves ⟶ Cylindrical power needs correction.

3. Fincham's test :

Uses	Procedure	Observation & Inference
Differentiate b/w cataract and glaucoma (Both present with halos around light)	Stenopic slit rotated in front of eye	• Halos remain intact : Indicative of ACG. • Halos appear broken ⟶ Dx : Cataract
Determine median for optical iridectomy	Vision compromised d/t corneal opacity. ↓ SS rotated to determine meridian least affected by corneal opacity.	Iridectomy performed along meridian

----- Active space -----

Note :

Empirical meridian for iridectomy → Inferonasal → For patients involved in near work.
→ Superotemporal → For patients involved in far work.

Amaurosis

01:06:49

Definition : Complete loss of vision in one/both eyes without any organic cause.
(No signs on ocular examination)

Note : Amblyopia → Partial LOV.

TYPES

Bilateral amaurosis :

Causes :

- Acute nephritis.
- Uremic amaurosis :
  - B/L complete LOV.
  - Pupillary reflexes intact as only higher visual centres affected.

Amaurosis Fugax :

Transient Complete LOV.

Causes :

- Thrombo embolic episodes (strokes) → m/c cause.
- CRAO (Central retinal artery occlusion).
- Papilloedema.

Aphakia

01:12:10

Absence of lens.

Causes :

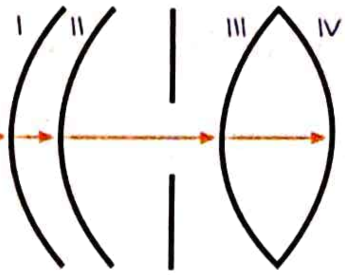
- Surgical.
- Traumatic.
- Congenital.

SYMPTOMS

- LOV for far and near vision (No accommodation).
- Refractory error : Hypermetropia (Low power of eye).
- AC deeper : Iris moves posteriorly d/t absence of lens.
- Irido-donesis (Trembling iris) : D/t lack of support posteriorly.
- Purkinje images : 2 visible.

Note : Purkinje images

- Beam of light
  when cast on eye :
  Reflects, forming purkinje images.



- Normal : 4 purkinje images.
  I : Anterior surface of cornea
  II : Posterior surface of cornea } Convex surface → Erect image.
  III : Anterior surface of lens
  IV : Posterior surface of lens → Concave surface → Inverted image.

TREATMENT

a. IOL implantation : Treatment of choice.

b. Soft contact lens

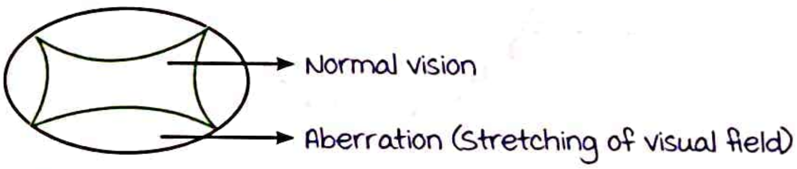
c. Spectacles :

- Spherical convex (+) power.
- maximum possible +10 D to +13 D : Heavy, large lens.

Side effects of high power spectacles :

3. magnification : upto 20% magnification (For +10 D spectacles)
   ↓
   Abberant perception of distance of objects
   ↓
   Objects appear nearer.

4. Cosmetic side effect : Eyes look large (Frog eyes).
5. Spherical aberration : Pin cushion defect.



Pin cushion defect

6. Prismatic abberation : Jack in box phenomenon/Roving ring scotoma.
   - mechanism : ↑Thickness of lens
     ↓
     Edges behave as a prism
     ↓
     multiple refractions occurring near edges
     ↓
     - Only central clear vision.
     - Peripheral vision compromised.

----- Active space -----

ANATOMY AND INVESTIGATIONS OF RETINA

00:01:33

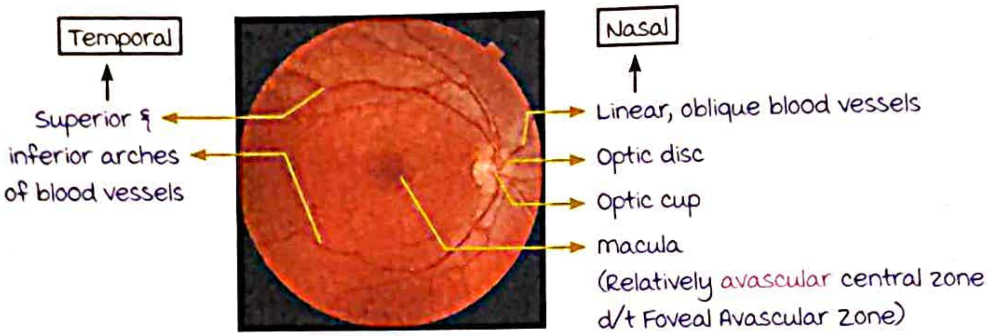
Anatomy

Colour of retina : Transparent.

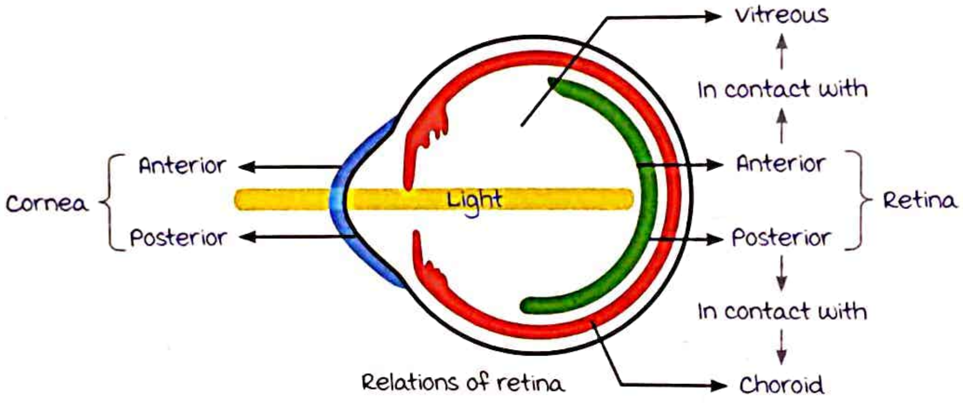
Number of layers : 10.

Gross anatomy :

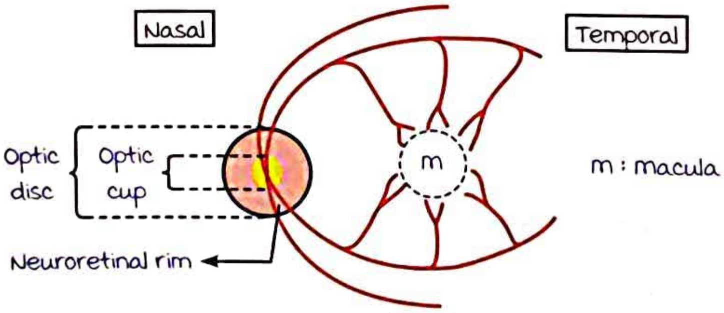
- Optic disc.
- macula
  - For central vision; Central part : Fovea.
  - Location : Temporal to optic disc.
- Ora serrata : Anterior termination of retina.
- Part of 'fundus' (View of all structures in posterior segment).


Temporal
Superior & inferior arches of blood vessels
Nasal
Linear, oblique blood vessels
Optic disc
Optic cup
macula
(Relatively avascular central zone d/t Foveal Avascular Zone)

Normal fundus


Vitreous
In contact with
Anterior
Retina
Posterior
In contact with
Choroid
Cornea
Anterior
Posterior
Light

Relations of retina

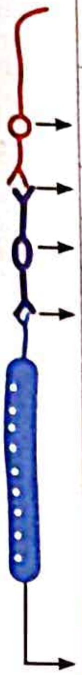

Nasal
Temporal
Optic disc
Optic cup
m
m : macula
Neuroretinal rim

Normal fundus

Layers of Retina :

----- Active space -----

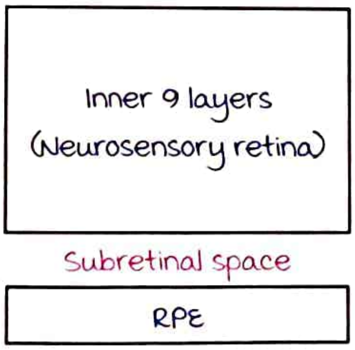
Ten layers : mnemonic → ING [IOP]$_3$ (Innermost to outermost).

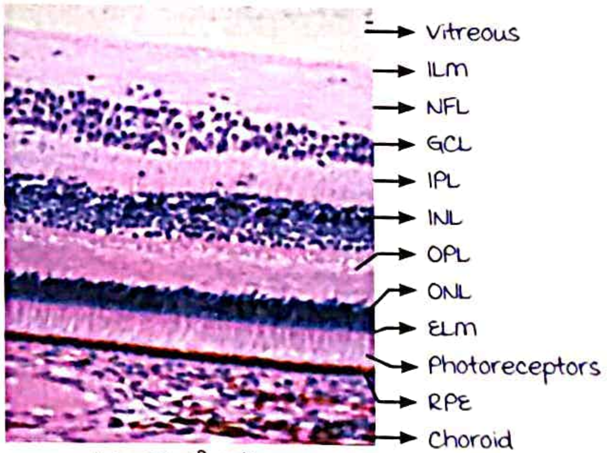


Layer	Feature
Internal limiting membrane (ILM)	• True basement membrane. • Formed by footplates of muller cells (Structural supporting cells).
Nerve fiber layer (NFL)	• Forms optic nerve (Collection of axons of ganglion cells). • 3rd order neurons.
Ganglion cell layer (GCL)	• Contains ganglion cells : 3rd order neurons. • Initial site of generation of action potential.
Inner plexiform layer (IPL)	Synapse between ganglion cell & bipolar cells.
Inner nuclear layer (INL)	Contains bipolar cells : 2nd order neurons.
Outer plexiform layer (OPL)	• Synapse between bipolar cells & photoreceptors. • Endothelial cells of capillaries → Form inner blood retinal barrier.
Outer nuclear layer (ONL)	
External limiting membrane (ELM)	Not a true basement membrane.
Photoreceptors	Rods & cones (1st order neurons). ↓ Receives light (After it passes through 8 layers). ↓ Generates local graded potential → Conducted back to nerve fiber layer.
Retinal Pigment epithelium (RPE)	• Phagocytic function. • Forms outer blood retinal barrier → Prevents collection of fluid in subretinal space.

Neuroglial cells (Structural supporting cells) in retina :

- Astrocytes
- muller cells
- microglia





Layers of retina

Blood supply of retina :

- Inner 6 layers : Central retinal artery
- Outer 4 layers : Short posterior ciliary artery
  (Also supplies choroid & posterior sclera).

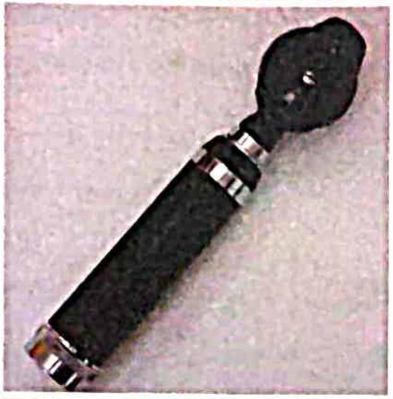
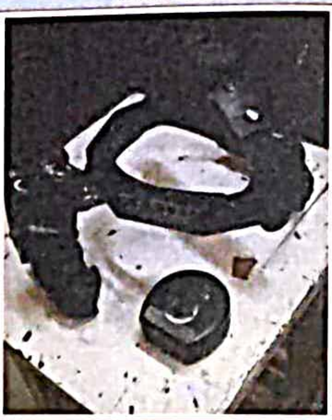
From ophthalmic artery
(Branch of internal carotid artery)

- macula : Supplied by central retinal artery ± Cilioretinal artery
  (Branch of short posterior ciliary artery)

00:35:09

Investigations

Ophthalmoscopy :

	Direct	Indirect
Instrument used		
magnification	15 times	3-5 times
External lens	Not used (Convex lens (+) within instrument)	Convex lens : Power → +20D
Image formed	Virtual & erect	Real and Inverted
Stereopsis/ Depth perception	Absent (monocular examination)	Present (Binocular examination)
Distance of examination	Close to patient's eye	An arm's length
Patient's position	Standing	Supine
Area visualized	• Smaller • Central fundus : Optic disc + macula • 2 disc diameters • 10° field of view	• Larger • Central + Peripheral fundus : Optic disc + macula + ora serrata • 8 disc diameters • 45° field of view
Evaluation with hazy media	Not possible	Possible
Indication	Pathology involving a specific part of retina. E.g. Glaucoma	Pathology involving any part of retina. E.g. Retinal detachment.

Fundus Fluorescein Angiography (FFA) :

Procedure :

5 ml of 10% solution Fluorescein dye (water-soluble orange coloured dye) injected into antecubital vein.

↓

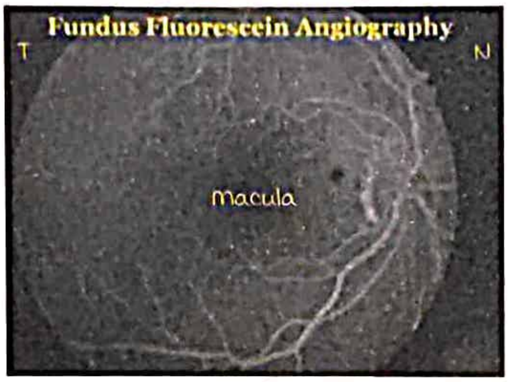
Blue light illumination.

↓

Excitation of fluorescein molecules at 520-530 nm.

↓

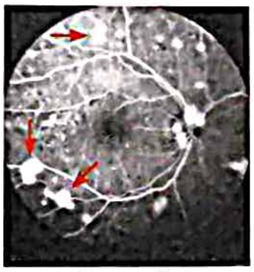
Blood vessels visualized.



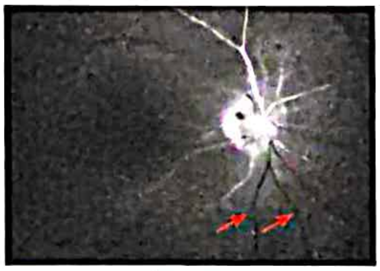
Normal FFA

Use : To diagnose

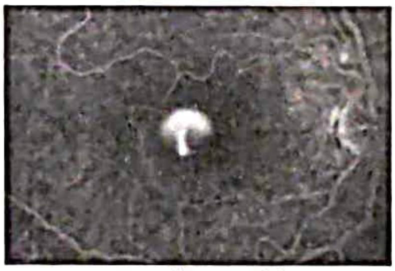
- Vascular disorders
- Macular disorders



Leakage of dye



Blocked vessel



Macula : Bright

Indo-Cyanine Green Angiography (ICGA) :

Dye used : Indocyanine green.

- 98% protein bound → Better retention.
- Fluoresces at 805 nm → Better penetration.

Use : For visualizing choroidal circulation.

C/I : Iodine hypersensitivity.

----- Active space -----

B-scan ultrasonography :

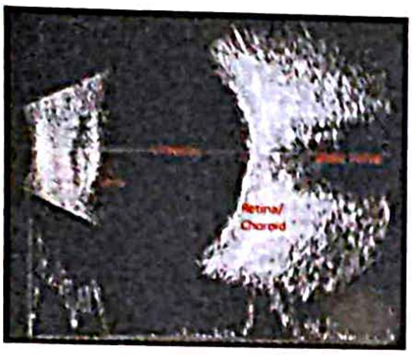
Brightness scan.

Mechanism : uses 7.5-10 MHz ultrasonic waves.

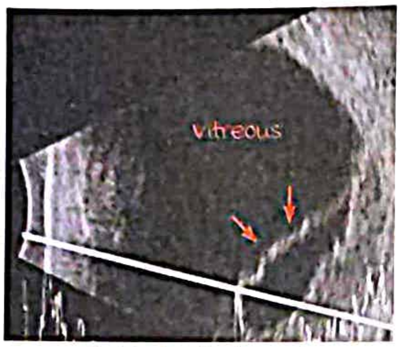
↓

Generates echoes
- → Hyperechoic area (Bright/white). E.g. → Retina (Anterior), → Choroid (Posterior)
- → Hypoechoic/anechoic areas (Dark/black). E.g. Vitreous.

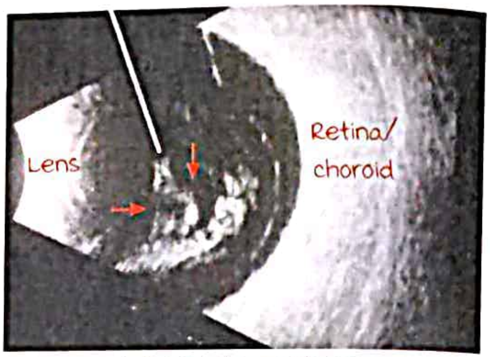
Indication : Patients with ocular media opacity → Difficult ophthalmoscopy.
(Corneal opacity/cataract)


Retina/
Choroid

Normal B-scan



Retinal detachment


Lens
Retina/
choroid

Vitreous hemorrhage

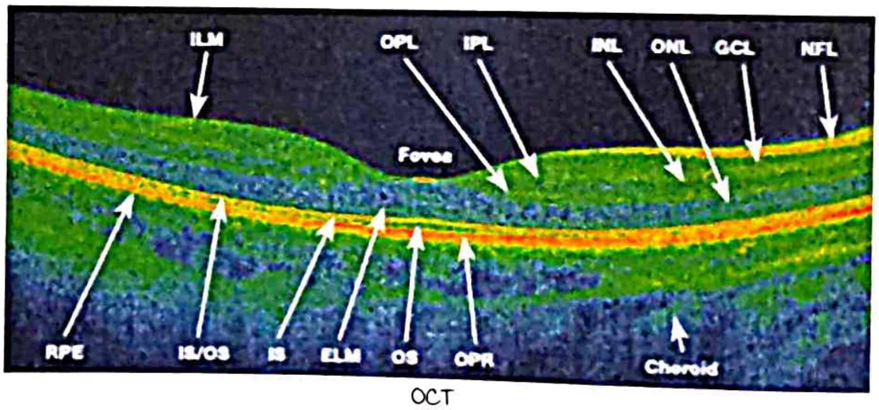
Optical Coherence Tomography (OCT) :

Principle : Low coherence interferometry

Optical biopsy → Visualizes microstructures of retina (To confirm diagnosis).

Landmarks :

- Fovea : Depressed d/t absence of ganglion cell layer.
- RPE : Red


ILM
OPL
IPL
INL
ONL
GCL
NFL
Fovea
RPE
IS/OS
IS
ELM
OS
OPR
Choroid

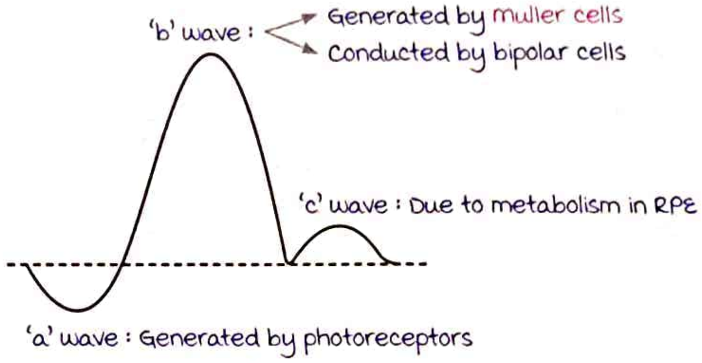
OCT

Electroretinography (ERG) :

- Graphical record of action potentials.
- Source : Panretina.

Special ERG :

1. Early Receptor Potential (ERP) } Generated by cones only (Function of macula)
2. Multifocal ERG } Generated by cones only (Function of macula)
3. Pattern ERG
    - Source : Ganglion cells → Represents optic nerve function
    - Use : To diagnose glaucoma.


'b' wave :
Generated by muller cells
Conducted by bipolar cells
'c' wave : Due to metabolism in RPE
'a' wave : Generated by photoreceptors

Waves in ERG

Electro-oculography (EOG) :

- Record of standing potential.
    - Produced d/t voltage difference across apical and basal segments of RPE cells.
    - Source :
        1. Photoreceptors } Outer retina
        2. RPE } Outer retina
- Use : To diagnose diseases of outer retina.
- Measured by Arden's ratio :

$$\frac{\text{Light peak (Highest value of EOG in well-lit conditions)}}{\text{Dark trough (Lowest value of EOG in dark)}} > 1.85 \text{ or } 185\% \text{ (Normal)}$$

RETINOBLASTOMA AND MACULAR DISORDERS

Retinal pathologies :

1. Retinoblastoma (Intraocular tumor).
2. macular diseases → Central loss of vision.
3. Fundus dystrophies < Disorders of cones / Disorders of rods
4. Vascular disorders.
5. Retinal detachment.

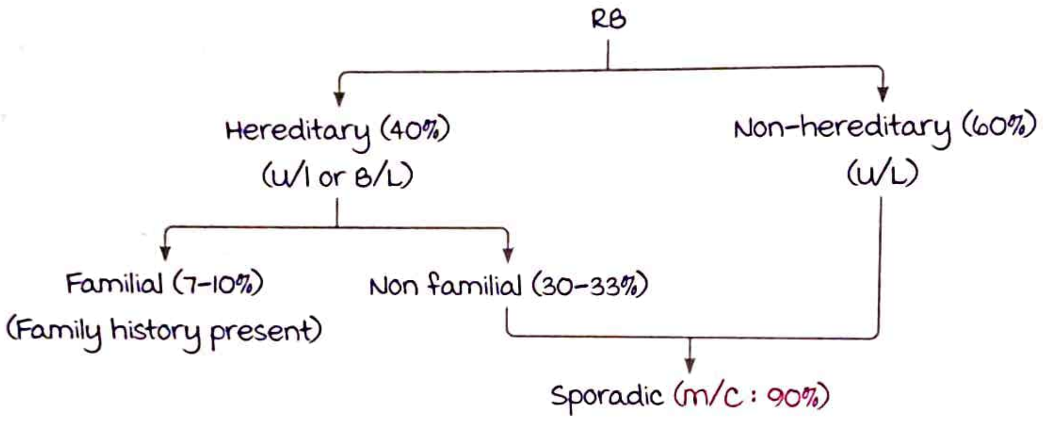
00:02:10

Retinoblastoma (RB)

m/c primary intraocular malignancy.

Incidence : 1 : 17,000

Inheritance :


RB
Hereditary (40%)
(U/l or B/L)
Non-hereditary (60%)
(U/L)
Familial (7-10%)
(Family history present)
Non familial (30-33%)
Sporadic (m/c : 90%)

Hereditary RB :

- mode of inheritance : AD
- Gene involved : RB-1 < Tumor suppressor gene / Location : Chr 13q14
- Knudson's 2-Hit hypothesis :

  RB < Genetypically : AD / Phenotypically : AR

  First mutation : Before birth (Rr)

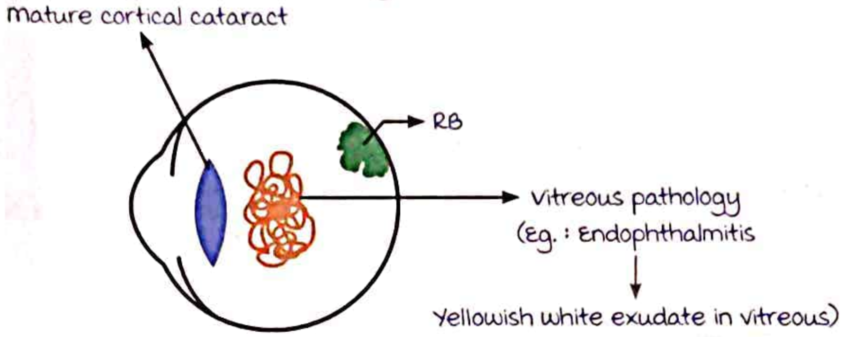
  ↓

  Second mutation : After birth (rr) → Loss of heterozygosity leads to malignant transformation & presentation.

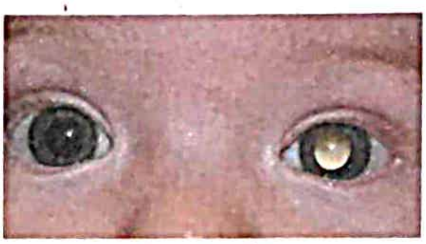
- Trilateral RB : B/L RB + Pinealoblastoma/PNET (Primitive neuroendocrine tumor
  ↓
  m/C malignancy associated with RB.
- m/C associated 2° malignancy : Osteosarcoma.

Clinical features :

1. Leucocoria : (m/C)
   - White pupillary reflex.
   - D/t loss of transparency of structures behind pupil.
   - Sign with various diagnoses.


Mature cortical cataract
RB
Vitreous pathology
(Eg. : Endophthalmitis
Yellowish white exudate in vitreous)

Causes of leucocoria



Leucocoria

m/C cause → U/L : RB ⟶ m/C presenting age : <3 years.
m/C cause → B/L : Cataract.

2. Squint (2nd m/C) ⟶ Esotropia.
3. 2° Glaucoma with buphthalmos (3rd m/C).
   ↓ D/t seeding from tumor.
   ↓ Enlarged eyeball.

Other features : D/t tumor invasion. Eg. : Iris invasion ⟶ Iritis.

Routes of Spread :

- Direct (m/C) : Through → Optic nerve (m/C) / Sclera
- Hematogenous.
- Lymphatic.

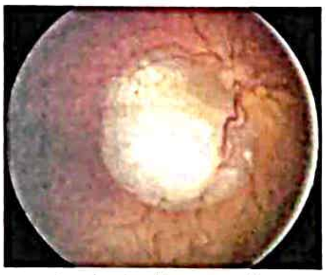
Growth pattern :

Endophytic : Grows into vitreous.

Exophytic : Grows towards choroid.

Retrolaminar :

- Worst prognosis.
- Spread via optic nerve to orbit & cortex ⟶ Intracranial metastases.



Endophytic growth

Investigations :

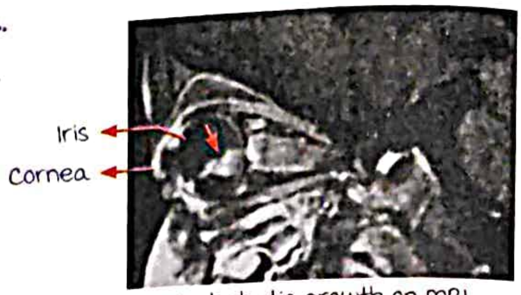
IOC : CT > X-ray (Since RB is m/c cause of intraocular calcifications)
↓
Can also detect intracranial metastases.

MRI : To assess for extracranial metastases.

LDH : ↑ed levels in aqueous humor.

Biopsy (Exicisional type) :

- Gold standard investigation.
- Sample : From optic nerve.
  ↓
  HPE done.

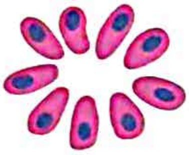


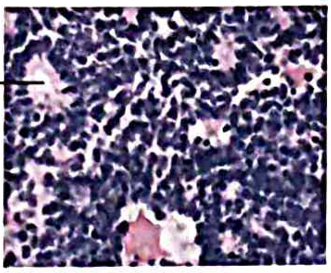
Endophytic growth on MRI

HPE features of biopsy :

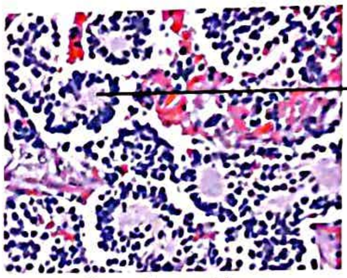
- Flexner Wintersteiner rosettes (Pathognomonic).
- Homer-Wright rosettes (Pseudorosette) : Also seen in → Neuroblastoma, → Ewing's sarcoma.
- Fleurettes.

Central empty space surrounded by cells with peripherally arranged nuclei.





Rosettes in RB



Pseudorosettes

Central entranglement of neural processes irregularly arranged cells & nuclei.

Treatment :

- Enucleation : TOC (Removal of whole eyeball with optic nerve).
- Chemotherapy : CEV → Carboplatin, Etoposide, Vincristine

Based on staging :

Stage	Treatment
G1	Cryotherapy/laser photocoagulation
G2	Carboplatin + Vincristine
G3	Carboplatin + Etoposide + Vincristine
G4	
G5	Enucleation

Grade 5 :

If any (+)

a. Tumour occupying >50% of total eye volume.

b. Vitreous hemorrhage (+).

c. Rubeosis iris (Neovascularisation of iris) (+).

Macular Disease

00:34:52

Early presentation : D/t central loss of vision.

If untreated → Chronic damage → Permanent loss of central vision.

Macula Function Tests :

With clear media :

- Visual acuity measurement.
- FFA.
- OCT.
- Photostress test.
- Amsler's grid (m/c).

With hazy media :

- maddox rod test.
- multifocal ERG.
- Laser interferometry.
- Potential Acuity meter (PAM).
- Entoptic phenomenon.

Photostress test :

On shining light on retina for 10s

↓

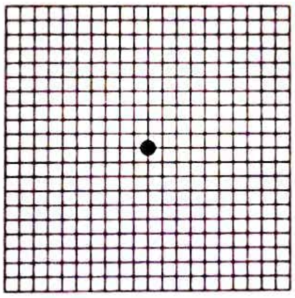
Time of recovery to within one line of pre-test vision

Normal → Optic nerve disease

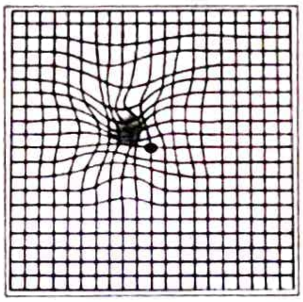
Delayed → Macular disease

Amsler's grid :

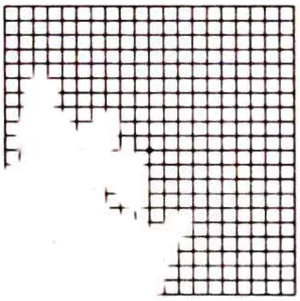
- Square chart with 400 5x5 mm squares.
- Differentiates macular disease from optic nerve disease.



Normal



In macular disease : wavy lines d/t change in contour of retina.



In optic nerve disease : Parts of lines missing.

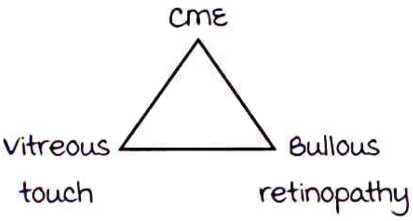
----- Active space -----

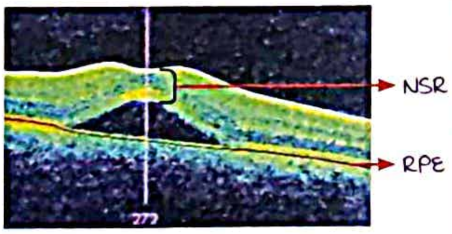
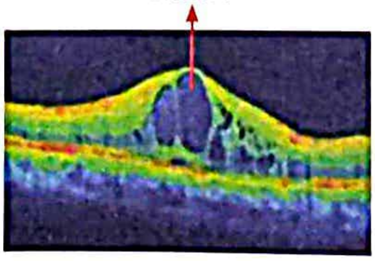
CENTRAL SEROUS RETINOPATHY VS CYSTOID MACULAR EDEMA

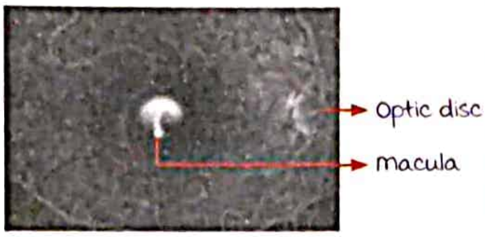
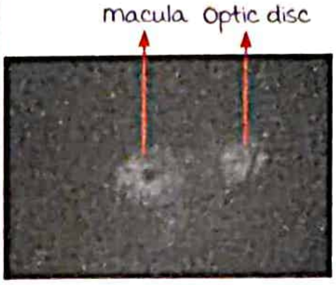
	Central Serous Retinopathy (CSR)	Cystoid macular Edema (CME)
Pathology	Collection of fluid at macula in subretinal space d/t defect in RPE.	Collection of fluid at macula in outer plexiform layer (6th layer).
Risk factors	• males (25-55 years). • Type A personality (↑stress). • Steroids. • Cushing's syndrome. • H.pylori infection	mnemonic : RUN for PRIDE • Retinitis pigmentosa. • Uveitis. • Niacin/nicotine. • Prostaglandin analogues. • Central Retinal vein occlusion. • Irvin Gass syndrome. • Diabetic retinopathy. • Epinephrine in aphakia.
Clinical features	1. Loss of central vision. 2. metamorphopsia (Distorted image).	

Irvin Gass syndrome :

Complication of cataract surgery.



	CSR	CME
OCT	Loss of foveal depression.	
	Shallow neurosensory retinal detachment in chronic cases. 	Cystic spaces in OPL in chronic cases. 

	CSR	CME
FFA (Pattern of pooling/leakage of dye)	• Ink blot appearance. • Smoke stack or Umbrella appearance.  Optic disc macula	Flower petal/petalloid appearance  macula Optic disc
Treatment	Self-resolving. Steroid : C/I. • CSR persistent > 4 months or • Recurrent or • Visual field defect in other eye ↓ a. Laser photocoagulation b. Photodynamic therapy : using Verteporfin ↓ On shining light of 689 nm, drug acts.	Steroids (mainstay)

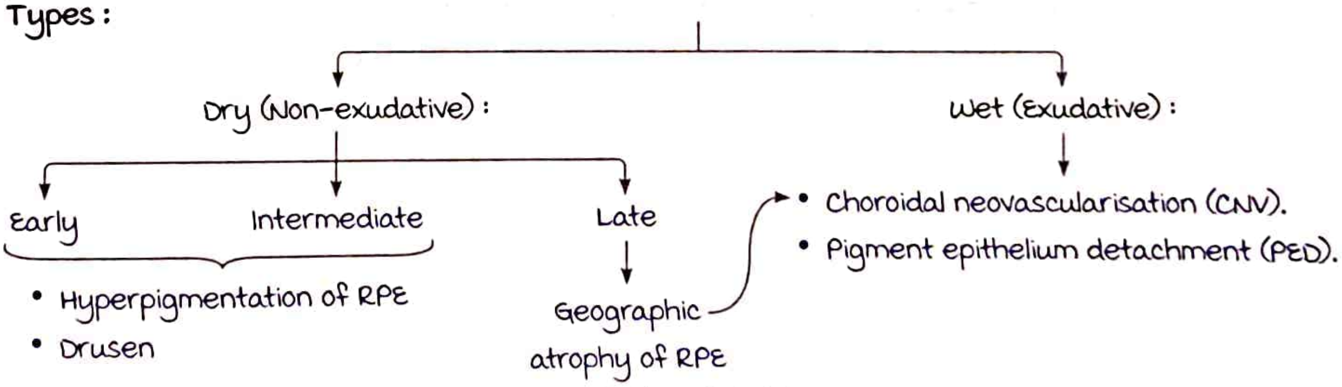
Age Related Macular Degeneration (ARMD)

01:01:12

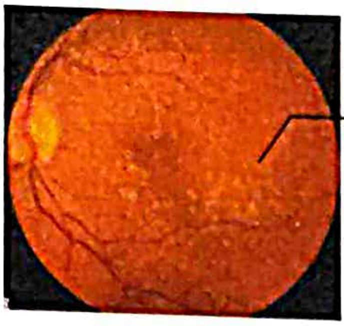
Risk factors :

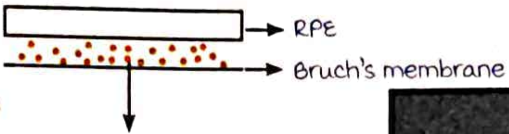
- Age >50 years.
- Smoking.
- Blue iris.
- High fat diet.

Types :


Dry (Non-exudative) :
Wet (Exudative) :
Early
Intermediate
Late
• Hyperpigmentation of RPE
• Drusen
Geographic atrophy of RPE
• Choroidal neovascularisation (CNV).
• Pigment epithelium detachment (PED).

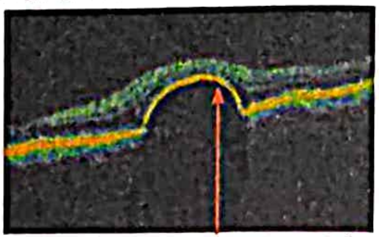
----- Active space -----





Drusen :

Extracellular eosinophilic deposits between RPE (Outermost layer of retina) and Bruch's membrane (innermost layer of choroid)



Pigment epithelium detachment (PED)

Choroidal neovascularisation :

Pathogenesis :

Damage to RPE & Bruch's membrane

↓

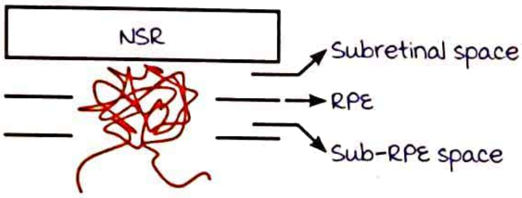
Vessels grow into subretinal space leading to hemorrhage

↓

Forms fibrovascular complex k/a CNVM (Choroidal Neovascular membrane)

→ Classical CNVM → IOC : FFA (In subretinal space)

→ Occult CNVM → IOC : ICGA (In sub-RPE space)



Treatment :

Dry ARMD	Wet ARMD
Antioxidants (AREDS-2 study) Fixed dose combination of : Vitamin C & E + Zinc & copper + Lutein & Zea-xanthin (Carotenoids)	a. In subfoveal CNVM : • Anti-VEGF drugs : TOC 1. Bevacizumab } Inhibits VEGFA isoform 2. Ranibizumab } Inhibits VEGFA isoform 3. Aflibercept : Soluble decoy receptor for VEGF 4. Brolucizumab • Photodynamic therapy
	b. In extrafoveal CNVM : Laser photocoagulation

Other Macular Disorders

01:15:31

----- Active space -----

Epiretinal membrane : AKA cellophane maculopathy.

Angioid streaks :

Find crack in Bruch's membrane.

Causes : PEPSI

- Pseudoxanthoma elasticum
- Ehler Danlos syndrome
- Paget's disease
- Sickle cell disease
- Idiopathic

Hypotony maculopathy :

D/t IOP <5 mmHg

Causes :

- Excessive drainage after glaucoma surgery.
- Retinal detachment.
- Penetrating trauma.
- Chronic uveitis.

----- Active space -----

DYSTROPHIES OF FUNDUS : RETINITIS PIGMENTOSA AND OTHERS

Affects photoreceptor retinal pigment epithelium (RPE) : Choriocapillaris complex → Degeneration of rods & cones.

Physiology of rods & cones :

	Rods	Cones
Vision	Night (scotopic) vision.	Day color (photopic) vision.
	Peripheral vision : D/t max concentration in perifoveal area.	Central vision : D/t max concentration at fovea.
Defect	Night blindness/Nyctalopia.	Color/day blindness/Hemaralopia/
	Peripheral loss of vision.	Central scotoma.
Causes	Of nyctalopia: mnemonic : OMG!(CRAP)$_2$ • Oguchi disease. • myopia. • Gyrate atrophy. • Congenital stationary night blindness. • Catarct : Cortical. • Retinal detahcment. • Retinitis pigmentosa. • Albinpunctatus : Fundus. • Vitamin A deficiency. • Phenothiazine toxicity. • Post-refractive surgery.	Of hemaralopia : mnemonic : C$_3$A$_3$T • Cone dytrophy. • Central cataract (nuclear & posterior subcapsular). • Cohen syndrome. • Achromatopsia. • Aniridia. • Albinism. • Trimethadione.

Retinitis Pigmentosa

00:07:35

m/c hereditary fundus dystrophy.

Inheritance :

- Autosomal recessive : m/c mode of inheritance.
- Autosomal dominant : Best prognosis.
- X-linked recessive : Worst prognosis.

Pathophysiology : Rod-cone dystrophy (rods affected first)

CLINICAL FEATURES

Symptoms :

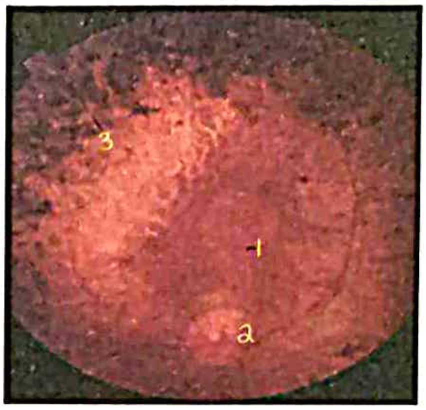
- Nyctalopia.
- Peripheral visual field loss.
  - mid-peripheral superior scotoma → Ring/doughnut scotoma → Tubular/ tunnel vision.

Signs :

1. Arteriolar attenuation.

Triad of
signs

2. Bony spicule (perivascular) pigmentation.
3. Waxy, pale optic disc d/t consecutive optic atrophy.



Fundoscopy

4. Tapetal reflex : Shining reflex at macula.
   - Seen in female carriers of XLR inheritance.

MANAGEMENT

Investigation :

Electroretinography (ERG) : IOC.

Treatment :

Not available in India.

Argus II retinal prosthesis :

- Restores central 20° vision.
- Done in complete loss of vision.

Voretigene neparvovec :

- Used in biallelic RPE-65 gene mutation.
- Adeno-associated virus vector based gene therapy.

SYSTEMIC ASSOCIATIONS

- Laurence moon Bell syndrome (m/c) : RP + intellectual disability + obesity+ polydactyly.
- Usher's syndrome : RP + Deafness.
- Refsum disease : RP + Cerebellar ataxia + Polyneuropathy.
- Bassen Kornzweig syndrome : RP + abetalipoproteinemia + acanthocytosis (spur cells).

Best's Macular Dystrophy

00:23:17

Autosomal dominant.

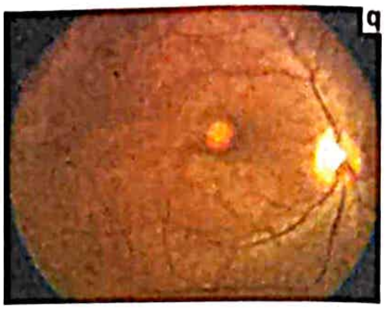
CLINICAL FEATURES

Symptoms :

- Hemaralopia.
- Central scotoma.

Signs :

- Lipofuscin accumulation.
- Fundus : Vitelliform lesion (egg yolk appearance)
- Pseudohypopyon : Collection of fluid in anterior chamber.
    - Vitelliruptive stage.



Vitelliform lesion

MANAGEMENT

Investigation :

- EOG : IOC
    - Arden's ratio : <1.5 or 150% (Normal : >1.85).
- ERG : Normal.

Stargardt's Disease

00:28:10

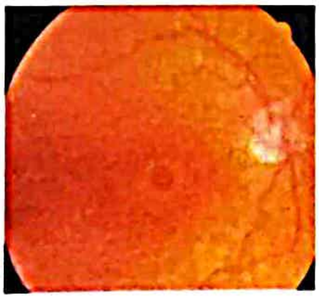
- Autosomal recessive.
- m/c juvenile macular dystrophy.

CLINICAL FEATURES

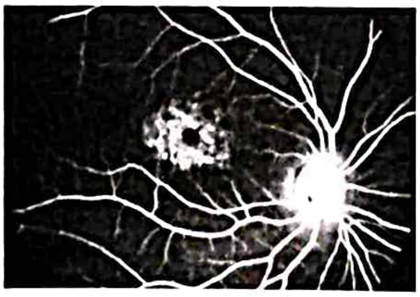
Symptoms :

- Hemaralopia.
- Central scotoma.

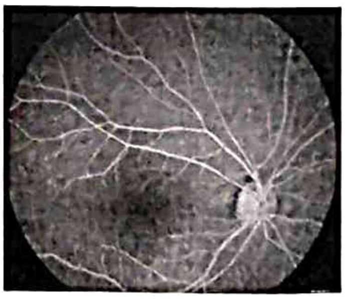
Signs :



Bull's eye maculopathy



Dark choroid/ Silent choroid sign



Normal FFA : Background flouresecence d/t dye in choroidal vessels.

Note : Chloroquine toxicity

1. Pre-maculopathy (reversible) : Normal vision + Central scotoma.
2. maculopathy : Bull's eye lesion.

- Seen if:
  - Daily dose : >3.5mg/kg or 250mg.
  - Cumulative dose : >460 g.
  - Duration of R_x : >5yrs.
  - Renal/liver dysfunction.
- Clinical features :
  - Diminished visual acuity.
  - Irreversible.

3. Vortex keratopathy.

Congenital Night Blindness Disorder

00:36:10

Aka Infantile onset nyctalopia.

Causes :

Causes	ERG	Fundus
Congenital stationary night blindness (CSNB)	Negative	Normal
Oguchi disease	Normal	Abnormal
Enhanced S cone syndrome	Negative	
Fundus albipunctatus	Absent rod responses	

Oguchi disease :

- Damage in conduction (No damage to rod cells).
- mizuo- Nakamura phenomenon :
  - Golden yellow fundus in bright conditions.
  - Dark adapted retina in dark conditions.

Miscellaneous

00:41:09

X-LINKED RETINOSCHISIS

- Split in retina between nerve fibre layer & rest of retina.
- O/E : Bicycle wheel maculopathy.

STICKLER'S SYNDROME

m/c inherited cause of RD in children.

RETINAL VASCULAR DISORDERS : PART 1

Diabetic Retinopathy

00:01:42

RISK FACTORS

- Duration of diabetes $\propto$ risk.
  - Time of screening → Type 1 : After 5yrs; → Type 2 : As soon as possible
- ↑ HbA1c.
- Pregnancy.
- Hypertension & hyperlipidemia.
- Diabetic nephropathy.

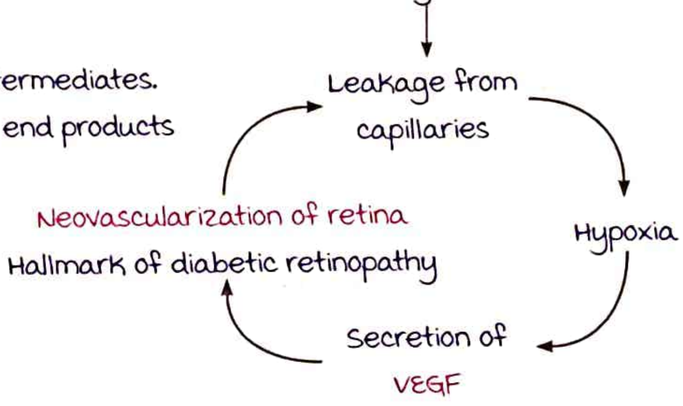
PATHOGENESIS

Biochemical Changes : → Anatomical vascular lesions :

- Protein Kinase C activation.
- Accumulation of :
  - ↑ Sorbitol.
  - Reactive oxygen intermediates.
  - Advanced glycation end products

Anatomical vascular lesions :

- Loss of pericytes of capillaries (Earliest change)


Leakage from capillaries
Hypoxia
Secretion of VEGF
Neovascularization of retina
Hallmark of diabetic retinopathy

Classification & Clinical Features of Diabetic Retinopathy

00:13:17

----- Active space -----

ETDRS (EARLY TREATMENT DIABETIC RETINOPATHY STUDY)

Non proliferative diabetic retinopathy (NPDR)

1. microaneurysms : Earliest sign seen in inner nuclear layers

O/E : Tiny red dots

FFA : Tiny hyperfluorescent dots

2. Hemorrhage :

- Flame shaped in superficial nerve fibre layer.
- Dot and blot hemorrhage in outer Plexiform layer.

FFA : Hyperfluorescent.

3. Exudates : Lipid deposits in outer plexiform layer

O/E : Yellowish irregular deposits

FFA : Hypofluorescent

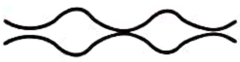
4. Cotton wool spots (soft exudates) : Collections of neuronal debris in nerve fibre layer.

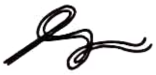
O/E : Large Yellowish deposits, regular & smooth edges

FFA : Hypofluorescent.

5. Venous tortuosity :

- Due to hypoxia
  - Beading.
  - Looping.





6. IRMA : Intra-retinal microvascular abnormalities

- Shunt vessels bypassing capillary bed
- Precursor to PDR.

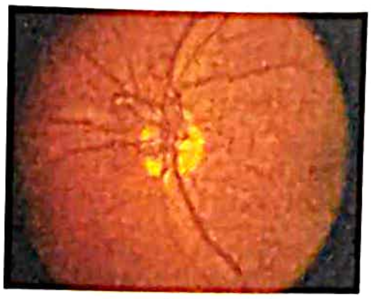
Proliferative diabetic retinopathy (PDR)

Neo-vascularization (NV) :

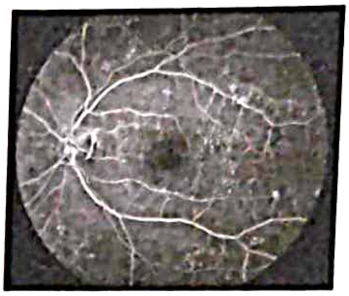
- O/E : Tortuous tuft of vessel.
- FFA : Hyperfluorescent.

NVD : NV at disc or within 1 disc diameter

NVE : NV elsewhere

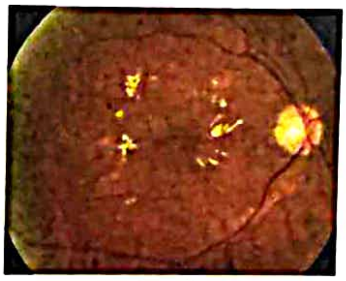


NVD

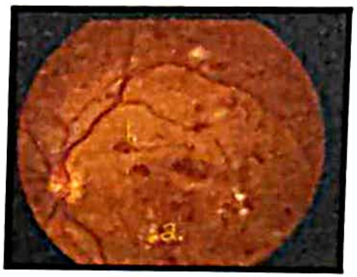


FFA imaging

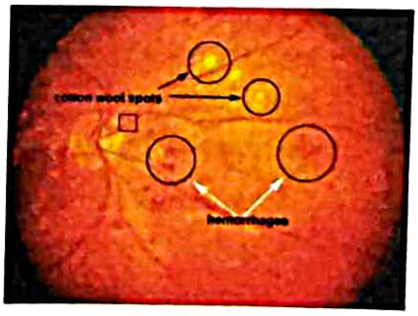
1. NVD.
2. microaneurysms.
3. Hyperfluorescent exudates.



Exudates



Hemorrhages (1) & exudates (2)



Cotton wool spots & hemorrhages

----- Active space -----

Treatment of Diabetic Retinopathy

00:32:48

NPDR

- Glycemic control.
- Follow-up

NPDR stage	Follow up in
mild	9-12 months
moderate	6 months
Severe	4 months
very severe	2 months

4-2-1 rule of NPDR :

- 4 quadrants hemorrhage.
- 2 quadrants venous beading.
- 1 quadrant IRMA :
  - Any 1 : severe NPDR.
  - ≥ 2 : very severe NPDR.

PDR

- Pan retinal photocoagulation : TOC
  - Argon green laser
  - multiple sittings.
  - S/E : Visual field defects.
- Anti VEGF drugs
  Ranibizumab, Bevacizumab, Aflibercept, Brolucizumab

Indication for treatment :

a. NVD > 1/3 disc area.
b. NVD with vitreous hemorrhage.
c. NVE > 1/2 disc area with vitreous hemorrhage.

Complications of Diabetic Retinopathy

00:42:07

1. Diabetic Macular Edema :

- D/t leakage from capillaries.
- m/c cause of loss of vision in NPDR (DR).

Rx :

1. Focal laser photocoagulation : TOC.
2. Intravitreal triamcinolone.
   Indications :
   - Ischemic maculopathy.
   - Pseudophakic patient.
   - Coexisting cardiovascular disease.
3. Anti VEGF drugs :
   - Indication : Centre involving edema within 300μm of + foveal thickness centre of macula > 250 μm

Indication for Rx :

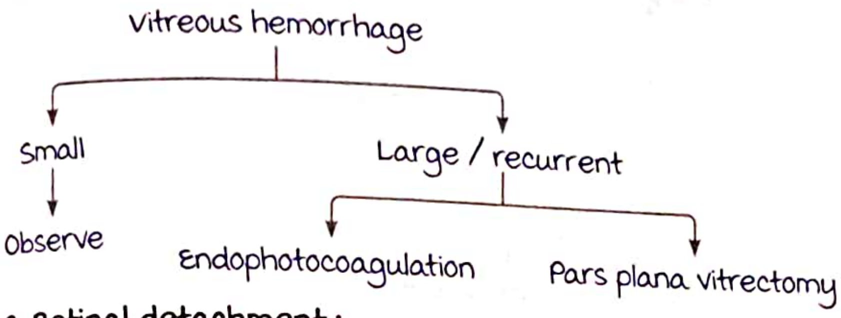
Clinically significant macular edema (CSME) :

- Retinal thickening within 500 μm of centre of macula.
- Exudates within 500 μm of centre of macula (retinal thickening anywhere).
- Retinal thickening or exudates (> 1 DD size) within 1DD (1500 μm) of centre of macula

2. Vitreous hemorrhage :

- Source of hemorrhage: Neovascularization of retina.
- m/c cause of loss of vision in PDR.
- m/c cause of vitreous hemorrhage
  - in adult : Diabetic retinopathy
  - in young adults (20-40 yrs) : Blunt trauma
- m/c cause of recurrent vitreous hemorrhage : Eale's disease

Rx :


Vitreous hemorrhage
Small
Observe
Large / recurrent
Endophotocoagulation
Pars plana vitrectomy

3. Retinal detachment :

m/c cause of blindness.

4. Rubeosis iridis :

- Neovascularisation of iris.
- D/t Oxygen demand by posterior segment on anterior segment.

----- Active space -----

RETINAL VASCULAR DISORDERS : PART 2

Hypertensive retinopathy

00:00:09

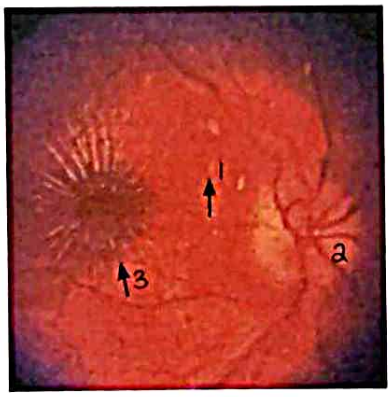
Vasoconstriction phase → Sclerotic phase (Atherosclerosis) → Exudative phase.

Clinical features :

Keith-Wagner classification :

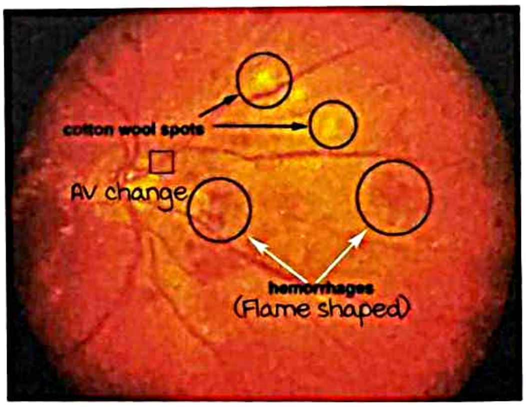
Grade	Characteristics
1	Arteriolar attenuation. • A : V (Diameter) ratio = 1:3 (Normal : 2:3).
2	Salus sign : Deflection of blood vessel.
3	• Bonnet sign : Banking of blood vessel. • Gunn sign : Tapering of blood vessel. • Flame shaped hemorrhages with cotton wool spots.
4	• Copper wiring of arterioles. • Silver wiring of arterioles. • Papilledema.

At AV crossings in veins



Grade 4 HTN retinopathy

1. Cotton wool spots
2. Swelling of optic disc
3. Exudates : macular star appearance : malignant HTN.



Grade 3 Hypertensive retinopathy

Central Retinal Artery Occlusion (CRAO)

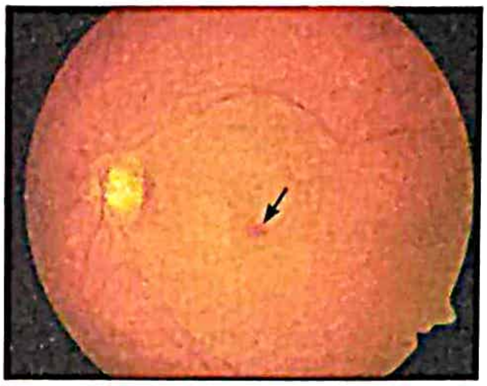
00:09:57

----- Active space -----

CAUSES

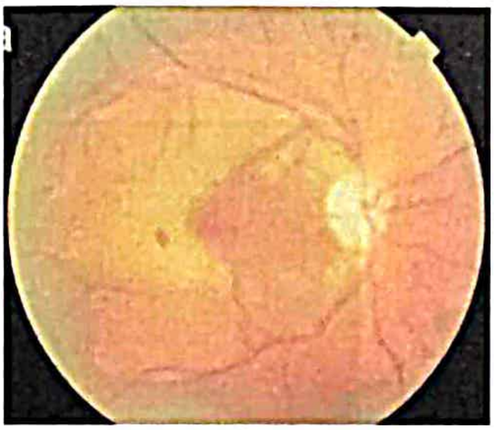
- Atherosclerosis : m/c.
- Emboli :
  - Hollenhorst plaque : Cholesterol emboli originating from bifurcation of CCA.
- Giant cell arteritis.

CLINICAL FEATURES

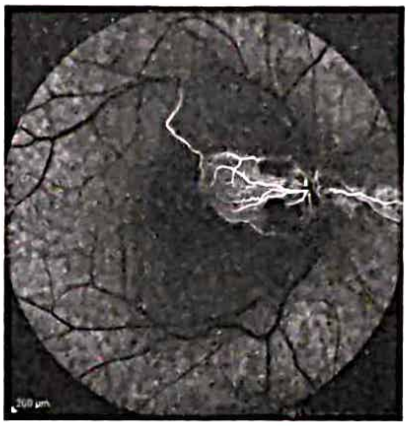


Cherry red spot

- Sudden, painless loss of vision.
- Optic atrophy (Consecutive).
- RAPD or Marcus Gunn pupil
- Fundus appearance :
  - Pale fundus : D/t retinal edema.
  - Cattle tracking fundus : D/t segmentation of blood column.
  - Cherry red spot at macula : D/t collection of fluid in ganglion cell layers (GCL) of retina except at foveola.



CRAO with macular cilioretinal artery sparring



Dye filling cilioretinal artery in FFA

Note : Cherry red spots at macula :

Mnemonic : Cherry trees never grow tall in sand, mud & grime.

- CRAO.
- Trauma-blunt.
- Niemann picks disease.
- Gangliosidosis Type I.
- Tay Sachs disease. } Gangliosidosis Type 2
- Sandhoffs disease. }
- Metachromatic leukodystrophy.
- Gaucher's disease : Type 2.

TREATMENT

Ocular emergency : Rx within 4 hrs.

24-48 hrs : Complete occlusion.

- Ocular massage : Dislodge emboli.
- Vasodilatation.
  - Sublingual isosorbide nitrate.
  - Carbogen : 95% O_2 + 5% CO_2 → Hypercarbia → $\underbrace{\uparrow PCO_2 \rightarrow \downarrow pH}_{\text{Respiratory acidosis}}$ → vasodilatation.
- ↓ IOP.
  - IV mannitol.
  - Paracentesis : Aspiration of aqueous.
- Rx of cause.

Central retinal vein occlusion (CRVO)

00:30:05

m/c cause : Hypertension.

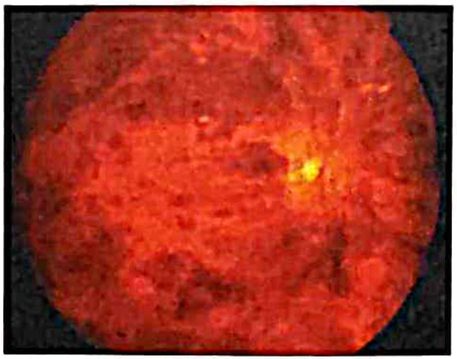
1. Non-ischemic stage :

D/t Stasis of blood → Hypoxia

D/t Stasis of blood → ↑ Vascular permeability → macular edema → Loss of vision

Rx :

- Intravitreal triamcinolone.
- 0.7mg Dexamethasone intra-vitreal implants (Geneva study).
- Anti VEGF drugs.



Tomato ketchup/splash appearance

2. Ischemic Stage :

Severe hypoxia → Capillary endothelium damage.

Clinical features :

- Severe flame shaped hemorrhages.
- Rubeosis iridis (Neovascularisation of iris) d/t Oxygen demand by posterior segment

↓

Blood leak into anterior chamber → Blocks trabecular meshwork → Neovascular glaucoma : AKA 100 day glaucoma : Glaucoma progression of 3 months in CRVO.

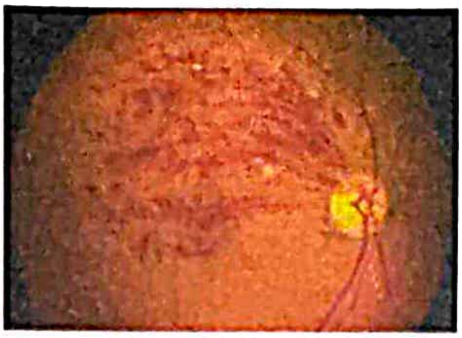
Rx : Panretinal photocoagulation.

Note :

m/c cause of neovascular glaucoma : Diabetic retinopathy.

BRANCHED RETINAL VEIN OCCLUSION (BRVO)

- Superior part of central retinal vein occluded.
- m/c site : AV crossings.
- m/c quadrant : Superotemporal.



BRVO

Retinopathy of prematurity (ROP)

00:41:53

Occurs in :

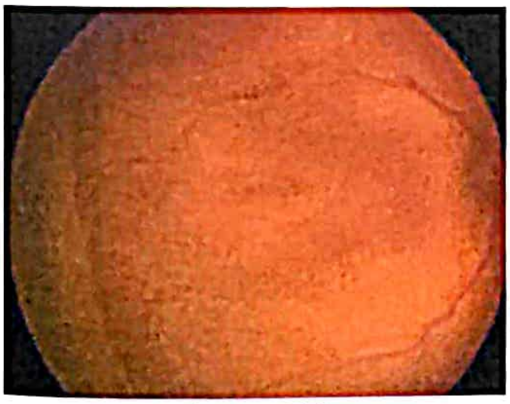
- Age at birth : ≤ 32 weeks.
- Birth weight : ≤ 1750 grams.

Age of screening :

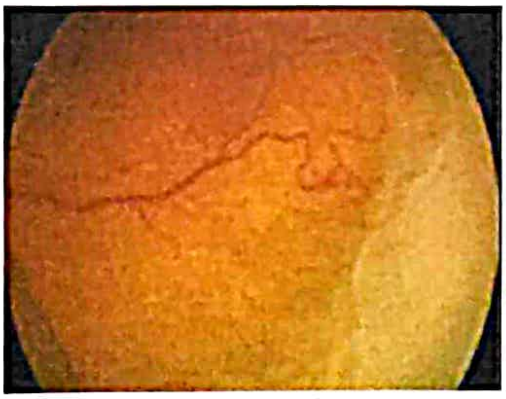
Criteria		Time of screening
Age at birth	Birth weight	
≥ 28 wks	≥ 1200 g	4 weeks after birth
< 28 wks	< 1200 g	2-3 weeks after birth

Stages :

Stages	Features
1	Demarcating line between vascularized retina & peripheral avascular retina
2	Ridge formation at demarcating line
3	Extra retinal neovascularization on the ridge moving into vitreous
4	Partial retinal detachment (RD)
5	Total RD



Stage 2 ROP

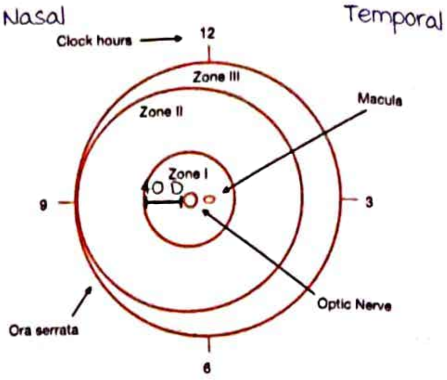


Stage 3 ROP

Treatment :

Laser photocoagulation :

- Indication : Pre-threshold ROP typer I (ETROP study).
  - Zone I + any stage + plus disease (Or)
  - Zone I + stage 3 (Or)
  - Zone 2 + stage 2/3 + plus disease (Or)
- Plus disease : Venous tortuosity & dilatation in ≥ 2 quadrants.


Nasal
Temporal
Clock hours
12
Zone III
Zone II
Macula
Zone I
O D
9
3
Optic Nerve
Ora serrata
6

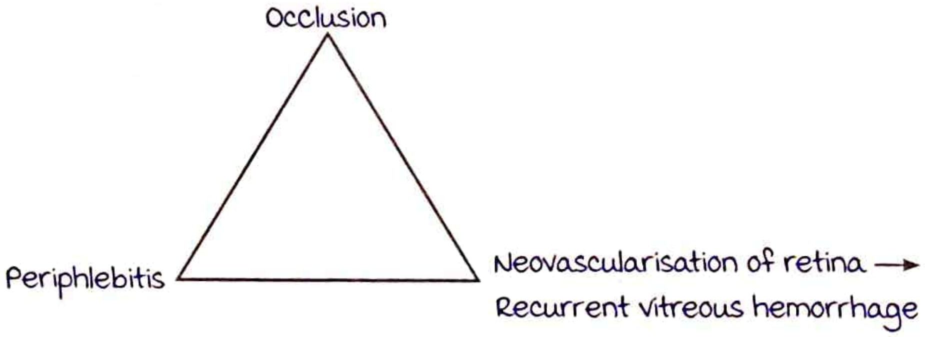
Zones of retina

Pars plana vitrectomy : If RD +.

Miscellaneous

00:53:52

EALES' DISEASE


Occlusion
Periphlebitis
Neovascularisation of retina →
Recurrent vitreous hemorrhage

Rx : Panretinal photocoagulation.

COATS' DISEASE

- Idiopathic retinal telengiectasia.
- m > F.
- Unilateral.

Clinical features :

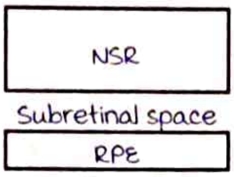
- Painless loss of vision.
- Strabismus.
- Leukocoria.
- Exudative retinal detachment.

RETINAL DETACHMENT

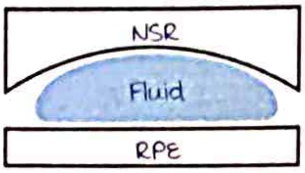
separation of neurosensory retina from retinal pigment epithelium and collection of subretinal fluid

Investigations :

- Indirect ophthalmoscopy : IOC.
- Optical coherence tomography (OCT).
- B-scan ultrasonography.


NSR
Subretinal space
RPE

Normal


NSR
Fluid
RPE

Retinal Detachment

Types :

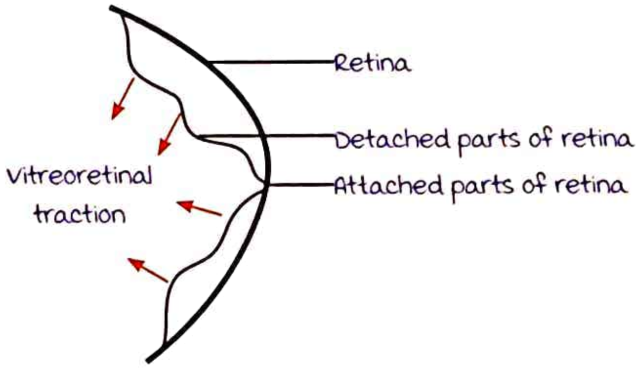
Retinal detachment (RD)
- Primary/Rhegmatogenous RD
- Secondary RD
  - Tractional
  - Exudative

Primary/Rhegmatogenous Retinal Detachment (RRD)

00:03:39

Pathogenesis :

Syneresis (Liquefaction of vitreous)
↓
Posterior vitreous detachment (PVD)
↓
Dynamic vitreoretinal traction from attached parts of vitreous.
↓
Retinal breaks develop.
↓
Fluid enters subretinal space.


Retina
Detached parts of retina
Attached parts of retina
Vitreoretinal traction

Causes :

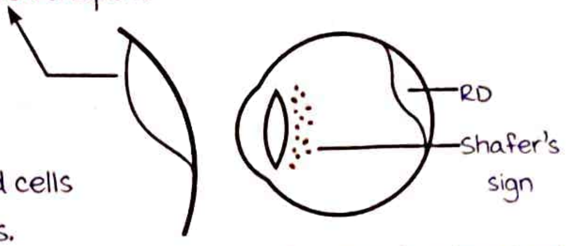
- Pathological myopia (m/c).
- Blunt trauma.
- Cataract surgery (Aphakia).
- Lattice degeneration of retina.

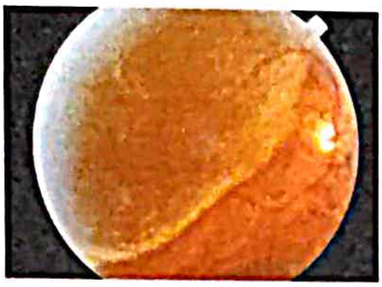
Symptoms :

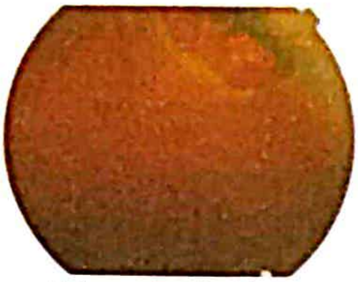
- Photophobia : Flashes of light (Stretching of nerve fibers → Ectopic neural impulses to brain).
- Floaters : Small black flying dots (D/t vitreal opacities).
- Curtain falling in front of eye.
- Loss of vision : Sudden & painless.

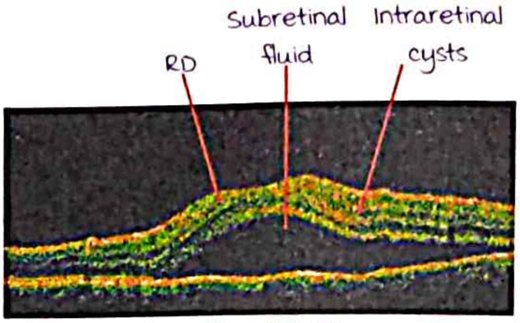
Signs :

1. Detached retina : Gray/opaque, convex shaped.
2. Extent of RD till ora serrata.
3. Shafer's sign.
   - Tobacco-dust appearance.
   - D/t deposition of brown pigmented cells at the anterior surface of vitreous.




RD


U shaped retinal tear



Longstanding RRD

M/C location : Superotemporal quadrant.

Lincoff rules : Rules to locate breaks in retina.

4. RAPD (Relative afferent pupillary defect).
5. In chronic RD :
   - Retinal atrophy.
   - Subretinal demarcation lines K/a High water marks (> 3 months).
   - Intraretinal cysts. (> 1 year).

Management :

Prophylaxis : Laser photocoagulation to seal breaks (usually superotemporal quadrant).

Treatment :

- Mobile retina → Tamponade
  - Internal
    1. Pneumatic retinoplexy
       - Uses SF_6 or C_3F_8 (Propane).
       - Done in superior breaks.
    2. Silicone oil.
       - Injected into vitreous cavity.
       - Removed after 8-12 weeks.
       - Complication : Hyperoleon → Oil leaks into anterior chamber and collects superiorly. (Inverse hypopyon).
  - External → Scleral buckling (Scleral weight pushes RPE anteriorly)
- Immobile retina → In proliferative vitreo-retinopathy (Causes membrane formation & scarring) → Vitrectomy + Subretinal fluid drainage

Note : Lincoff rules → Used to locate breaks in retina.

Secondary Retinal Detachment

00:25:06

----- Active space -----

	Tractional RD	Exudative RD
Pathology	Static vitreoretinal traction.	Exudation into subretinal space.
Etiology	• Diabetic retinopathy (m/c). • Retinopathy of prematurity. • Penetrating trauma. • Sickle cell retinopathy (Roth spots (+) : Hemorrhage with clear centre). O	• Choroidal melanoma (m/c). • Toxemia of pregnancy. • Coat's disease (Idiopathic retinal telengiectasia). • VKH syndrome • Central serous retinopathy.
Clinical Features	• Loss of vision : Gradual/ & painless. • Shape of RD : Concave. • Photopsia. • Floaters. • Extension of RD till ora serrata. } (-)	• Loss of vision : Sudden, painless. • Shape of RD : Convex. • Hallmark features : Shifting fluid (with shifting head position).
Treatment	Treat the cause.	

SPECIAL INVESTIGATIONS FOR CORNEA

Keratometry

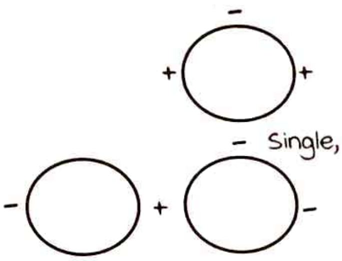
00:02:11

Principle : Anterior surface of cornea acts as a convex mirror.

- Size of image ∝ 1/curvature.
- Image seen as keratometric mires.

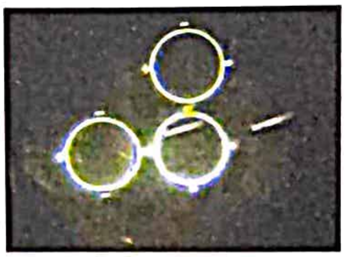
Keratometer

Correct positioning of mires :



Single, overlapped minus sign

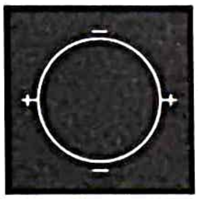
Single, overlapped plus sign



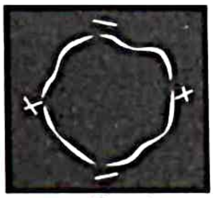
Keratometric mires

Uses :

1. Measure corneal curvature.
2. Diagnose astigmatism :
    - Horizontally oval mires : With the rule astigmatism.
    - Vertically oval mires : Against the rule astigmatism.
3. To ascertain proper fitting of contact lens.
4. To diagnose keratoconus (pulsating mires seen).



Good fit of lens



Steep fit of lens

Pachymetry

00:10:13

Measures corneal thickness.

Normal Corneal Thickness (CT) : 0.5 to 0.6 mm (0.54 mm) or 540µ.

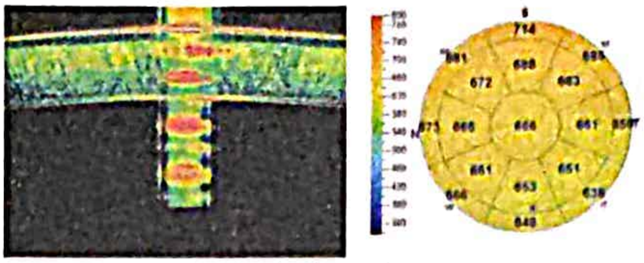
- CT is highest in the centre → Reduces towards periphery.

Uses :

1. Eligibility for LASIK :

- c/i if CT < 450μ.

2. IOP measurement : 1mm Hg IOP = 10 μm of CT.
   - CT ↑ → False high IOP
   - CT ↓ → False low IOP.



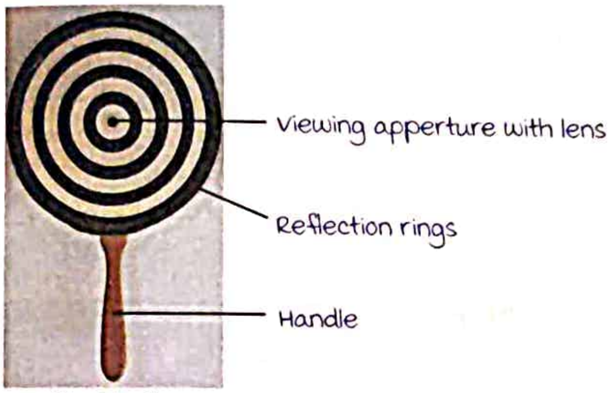
Pachymetry

Corneal topography 00:14:00

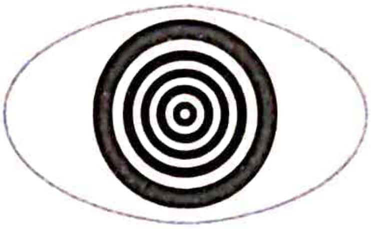
Examination of corneal surface.

methods used :

1. Placido disc :



Placido disc



Reflection of placido disc : Normal

2. Orbscan : uses slit scan imaging principle.
3. Pentacam :

   measures :

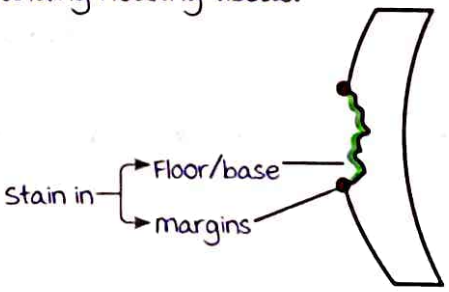
   a. CT.
   b. Corneal curvature.
   c. Anterior segment imaging.
   d. Topographic elevation maps of anterior surface.
   e. Topographic elevation maps of posterior surface.

Corneal vital staining 00:18:40

Used to visualize and distinguish ulcer from surrounding healthy tissue.

Fluorescein dye :

- Stains area of denuded epithelium (Floor/base of ulcer).
- Visualized under cobalt blue light → Green fluorescence.



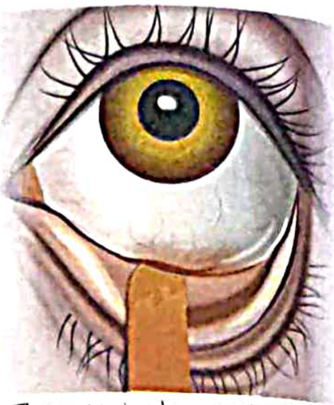
Other uses :

- Goldmann's applanation tonometry.
- Jones' dye disappearance test : Lacrimation assessment.
- Seidel's test (In penetrating trauma).

Rose Bengal dye :

- Stains devitalised/necrotic tissue (margins of ulcer).

Disadvantage : Can be toxic to healthy tissue.



Flourescein dye application

Lissamine green dye :

Stains devitalised/damaged tissue.

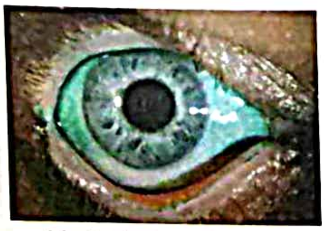
Advantage : Non-toxic to healthy ocular tissue.

Other uses :

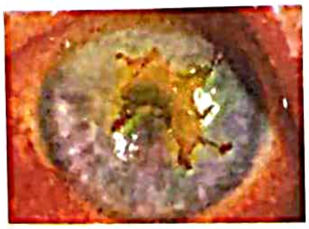
m/c used for diagnosis of dry eye by conjunctival staining.

Alcian blue dye :

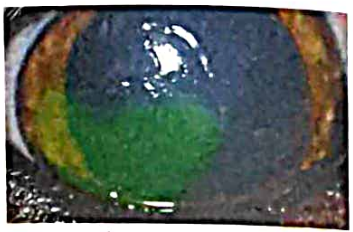
Stains
- mucin deposits
- Filaments.



Lissamine green dye



Rose bengal stain



Flourescein dye

Other corneal investigations

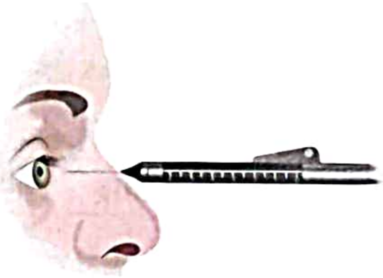
00:28:04

Confocal microscopy :

Non-invasive technique for in-vivo imaging of all layers of living cornea.

Corneal aesthesiometer :

Examines corneal sensations.



Corneal aesthesiometer

Instrument : Pen-like instrument with filament (6cm long).

- Length of filament illiciting corneal sensations $\propto$ corneal sensations $\propto$ 1/anaesthesia

CORNEAL ULCER AND KERATITIS

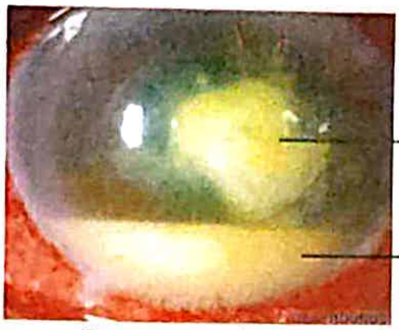
Discontinuity in corneal epithelium + Cellular infiltration & necrosis of underlying layers.

Bacterial Corneal Ulcer

00:02:20

ETIOLOGY

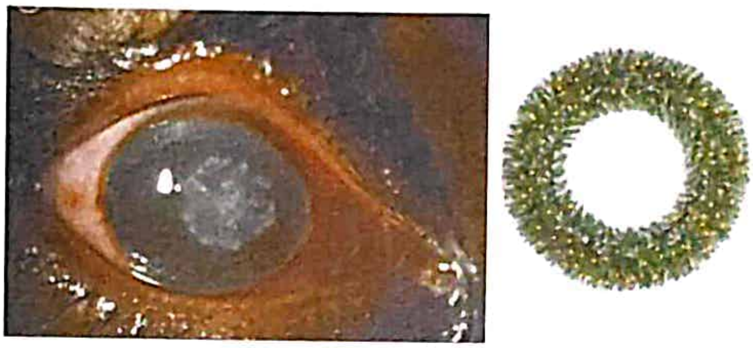
- Staphylococcus aureus : m/c worldwide.
- Pneumococcus : MC in India.
  - Ulcer serpens : Centre → Periphery in zigzag pattern.
  - Hypopyon corneal ulcer.
- Pseudomonas : m/c in Contact lens users.
  - Green discharge.
- Atypical mycobacteria : m/c in h/o LASIK.
- Nocardia : Wreath shaped/Pin head ulcer.



Pneumococcal ulcer

Bacteria invading intact epithelium :

- Neisseria : N. meningitidis, N. gonorrhoea.
- Corynebacterium diphtheria.
- Listeria.
- Shigella.
- Haemophilus aegyptius.

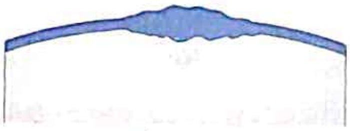


Wreath shaped ulcer

PATHOGENESIS

Stage 1–Initial stage :

Saucer shaped ulcer with overhanging margins.

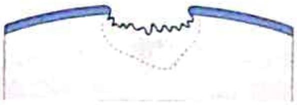


Stage 1

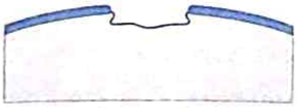
Stage 2–Progressive stage :

Infiltration by PMNs.

- Hypopyon.
- Keratouveitis.
  - Mobile : Liquid like pus.
  - Sterile.



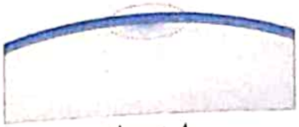
Stage 2



Stage 3

Stage 3-Regressive stage :
- Smooth floor & edges of ulcer.
- Vascularization + .
- ↓ Inflammatory response.

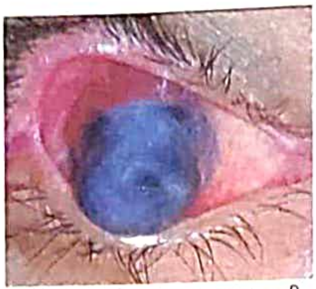
Stage 4-Scar/Cicatrization :
Permanent vision loss.

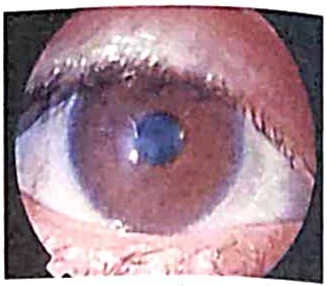

Stage 4

COMPLICATION

1. Ectatic cicatrix :
   - Thinning of Cornea.
   - Bulging of cornea d/t ↑ IOP (Temporary/permanent).

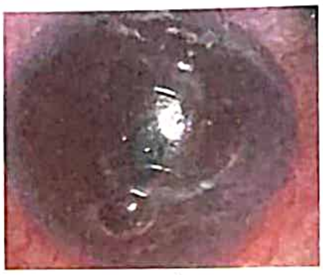
2. Anterior staphyloma :
Bulging of Cornea d/t thinning + incarceration of iris tissue.

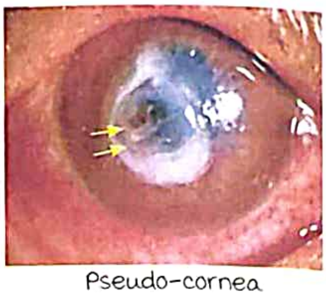

Lobulated Appearance of Anterior Staphyloma


Descemetocoele

3. Descemetocoele :
Herniation of Descemet's membrane through Corneal ulcer.

4. Perforation of cornea :
   - a. Aqueous leak → ↓ IOP.
   - b. Iris prolapse.
   - c. Pseudo-cornea : Corneal surface formed d/t plugging of perforation by iris tissue.


Iris Prolapse


Pseudo-cornea

5. Endophthalmitis/Panophthalmitis.

MANAGEMENT

Antibiotics :
- After gram staining & culture of ulcer base, scraping.
- Fortified antibiotics : Fortified Cephalozin 5 % + Fortified Tobramycin 1.3 %.

Atropine eye drops : Relieve ciliary spasms.

Impending perforation :
- Oral doxycycline.
- Soft Bandage Contact Lens (BCL).
- Cyanoacrylate glue.

Contraindications :
Steroids : D/t epithelial thinning.

Fungal Corneal Ulcer

00:30:00

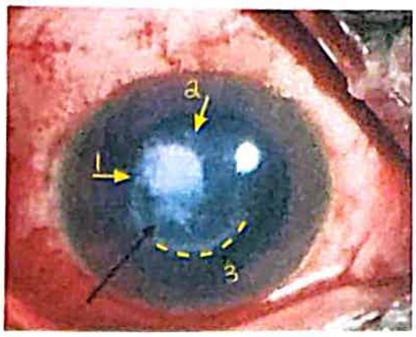
----- Active space -----

- m/c cause : Aspergillus (Filamentous fungi with septate hyphae).
  - H/O trauma with vegetative fungi.

Signs :

Out of proportion to symptoms.

- Dry looking ulcer. → 1. Feathery ulcer margin. 2. Satellite lesions. 3. Weasley Immune Ring : Antigen-antibody complexes.
- Hypopyon :
  - Thick, immobile.
  - Asterile.



Fungal Corneal ulcer

Treatment :

- Antibiotics :
  - Natamycin 5 % eye drops : DOC for Aspergillus fusarium.
  - Amphotericin B 0.15 % eye drops.
- Atropine : Supportive Rx.
- Steroids : C/I.

Acanthamoeba Keratitis

00:38:19

Risk factors :

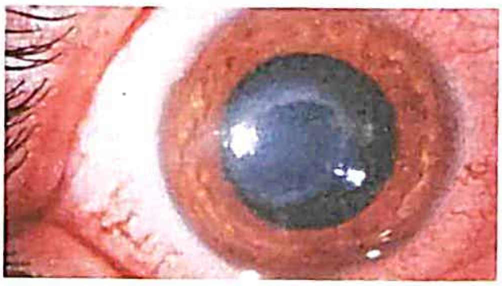
- Unhygienic practices in contact lens users.
  - Contaminated solution, washing in tap water, swimming pools.

Clinical features :

Symptoms out of proportion to signs.

Signs :

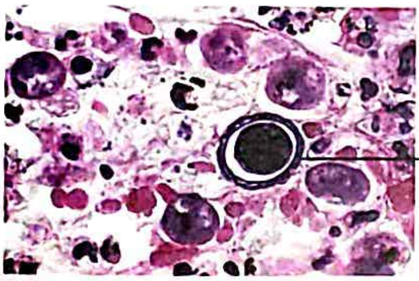
- Pseudodendritic keratitis.
- Radial keratoneuritis : Inflammation of nerves throughout cornea.
- Ring shaped ulcer with abscess : Pathognomonic.



Ring abscess

Investigation :

1. Gram staining :



2. Culture media :

Non- nutrient agar enriched with E. coli.

----- Active space -----

Treatment :

Drugs inhibiting membrane synthesis :

- PHMB 0.02% (Polyhexamethylene biguanide) : Doc.
- Chlorhexidine 0.02%.

Drugs inhibiting DNA synthesis :

Propamidine : m/c.

Viral Keratitis

00:44:58

HERPES SIMPLEX VIRUS (HSV)

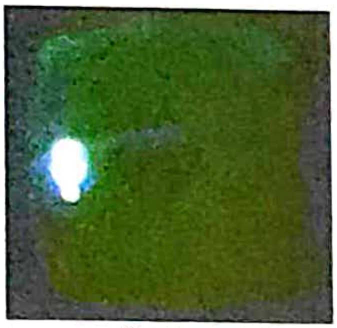
1° infection : Conjunctivitis.

Recurrent infection : Keratitis.

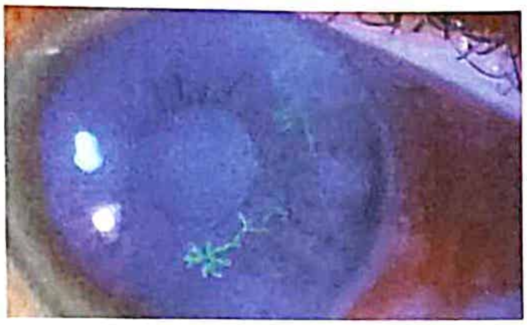
Clinical features :

a. Epithelial lesions : D/t active viral replication.

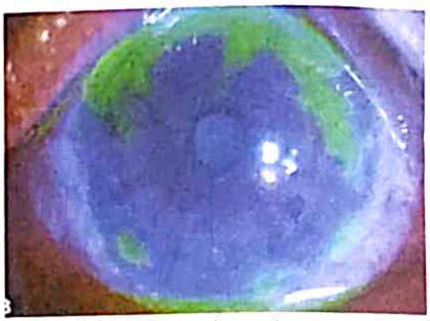
Fluorescence staining :



Superficial punctate (Dot like) Keratitis



Dendritic ulcer : Branching with terminal buds.



Geographic ulcer

Rx :

- 3% acyclovir eye ointment : 5 times/day x 10-14 days.
- Steroids : C/I.

Note : Other d/d's for Dendritic ulcer :

- Acanthamoeba Keratitis.
- HZV Keratitis.
- Tyrosinemia.
- Contact lens overuse.

b. Stromal Keratitis :

i. Immune Stromal Keratitis :

D/t type III hypersensitivity.

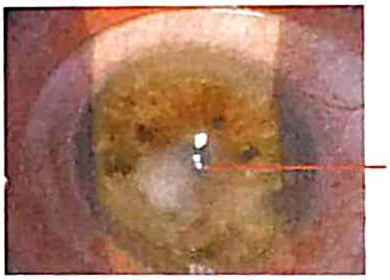
Occurs following epithelial Keratitis.

Rx : 3% Acyclovir ointment + Topical steroids.

ii. Necrotizing Stromal Keratitis :

most severe form of stromal Keratitis.

Rx : 3% Acyclovir ointment + Topical steroids.



Immune Stromal Keratitis

c. Endothelial lesion :

i. Disciform Keratitis :

- Disc shaped areas of deep stromal/endothelial edema.
- Type IV Hypersensitivity reaction.

Hallmark of HSV Keratitis :

Corneal anaesthesia → metaherpetic Keratitis/Neurotrophic ulcer : D/t 5th Cranial Nerve palsy.

HERPES ZOSTER OPHTHALMICUS/SHINGLES

- Affects dermatome supplied by ophthalmic branch of 5th cranial nerve.
- Hutchinson's sign : Rash involving tip of the nose → ↑ chance of Herpes zoster Ophthalmicus.

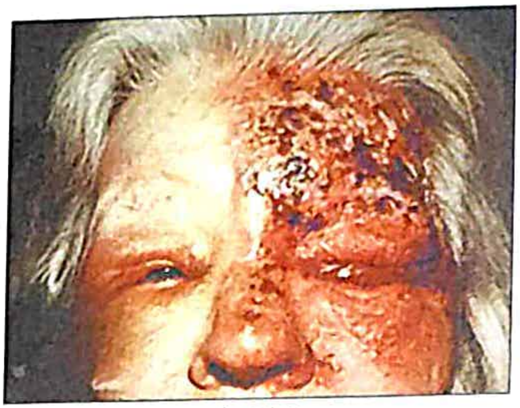
Clinical features :

1. Pseudodendritic ulcer : Branching pattern with no terminal buds.
2. Nummular Keratitis.
3. mucous plaque Keratitis : In chronic cases.
4. Stromal Keratitis.
5. Cranial nerve palsy : 3rd cranial nerve-m/c.
6. ScleroKeratitis : Least common finding.
7. Post-herpetic neuralgia :

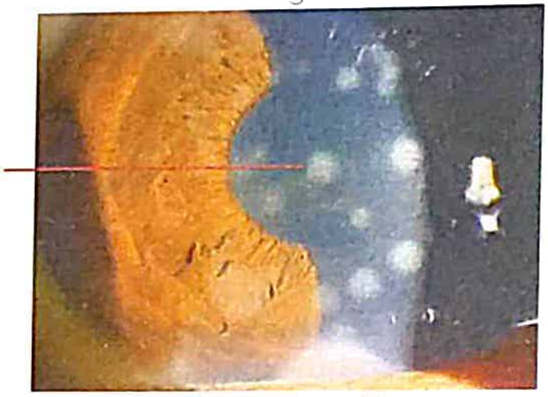
- Persistence of pain after 1 month of resolution.
- Worse at night.

Treatment :

T. Acyclovir 800mg x 5 times/day x 2 weeks.



Shingles



Coin shaped sub-epithelial lesions

Nummular Keratitis

CORNEAL DYSTROPHIES, KERATOCONUS AND MISCELLANEOUS DISORDERS

Corneal Dystrophies

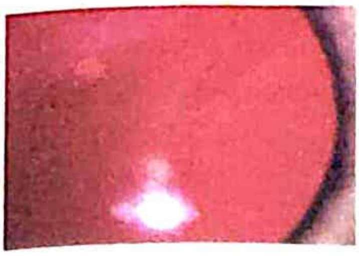
00:00:12

Features :

- Primary corneal disease.
- Present in 1st and 2nd decades.
- Non-inflammatory, progressive, B/L.
- Autosomal dominant inheritance.

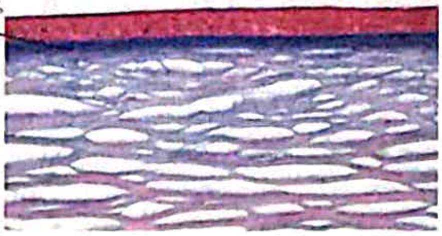
Disease	Feature	
1. Epithelial		
Cogan's epithelial basement membrane dystrophy/ map dot	m/c corneal dystrophy.	
meesmann epithelial dystrophy	Non-progressive, d/t keratin gene mutation.	
Lisch epithelial dystrophy	AD/XLD.	
2. Bowman layer		
Reis Buckler's dystrophy/CDBI/GCD III	Fish net.	
Schnyder Central Crystalline dystrophy	Corneal lipid metabolism disorder.	
Thiel Behnke dystrophy / GCD III	Honeycomb pattern of opacities.	
3. Stromal		
Lattice corneal dystrophy I/ Biber Haab Dimmer lattice dystrophy II/ Finnish	Neuropathy seen 70-90 years.	Amyloid deposit : Stained by Congo red
Lattice corneal dystrophy IIIA	AR, mulberry-like appearance.	
Gelatinous drop like dystrophy/Japanese	Hyaline deposit : Stained by masson Trichome stain.	
Granular corneal dystrophy I/ Groenaw granular corneal dystrophy II	Hyaline + Amyloid deposit	
macular dystrophy/Groenow II	AR, L/C corneal dystrophy	
Francois central cloudy dystrophy	Inborn error of keratin sulphate metabolism.	
4. Endothelial		
Fuch's endothelial dystrophy	Corneal guttata : wart-like excrescences.	
Posterior polymorphous corneal dystrophy	-	
Congenital hereditary endothelial dystrophy I Congenital hereditary endothelial dystrophy II	Both have AR inheritance and have perinatal onset.	

meesman epithelial dystrophy :



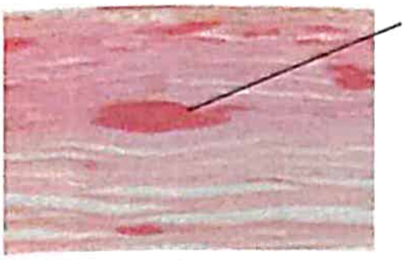
microcysts in corneal epithelium

macular dytrophy :

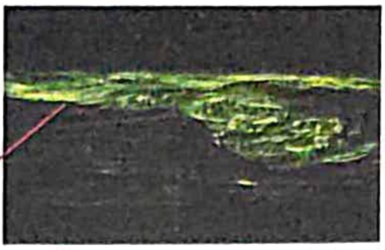

Acid mucopolysaccharidosis deposits

Alcian blue stain

Lattice Corneal dystrophy -Type I :

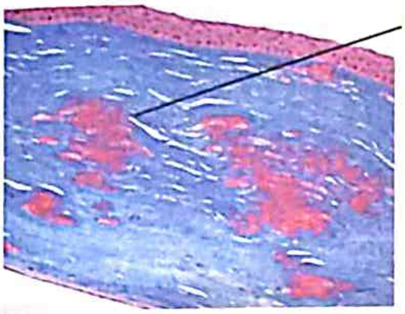

Amorphous deposit

PAS stain

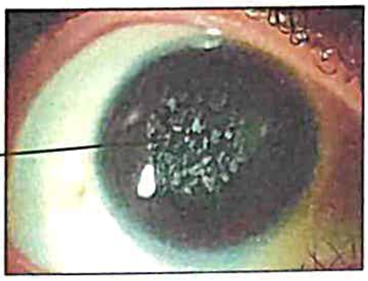

Apple green birefringence

Congo red stain

Granular dystrophy :


Hyaline deposits

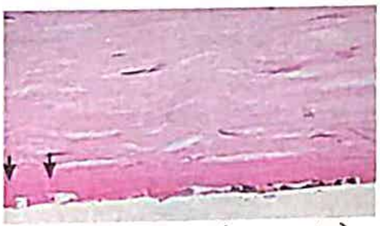
masson trichome stain


Separated by well-demarcated clear space

Bread crumb like granules

Fuch's endothelial dystrophy :

- mutation of COAxa gene.
- Descemet membrane + Endothelium damaged
- Wart-like excrescences on cornea



Corneal guttae (Diagnostic)

RECURRENT CORNEAL EROSIONS

Causes :

1. Cogan dystrophy.
2. Thiel Behnke dystrophy.
3. Reis Buckler dystrophy.
4. Lattice type I.

----- Active space -----

METABOLIC CAUSES OF DYSTROPHY

1. meesmann epithelial dystrophy.
2. macular dystrophy.
3. Schnyder central crystalline dystrophy.

TREATMENT OF CORNEAL DYSTROPHIES

If visual loss + : Keratoplasty/ Corneal transplantation.

Keratoconus

00:15:54

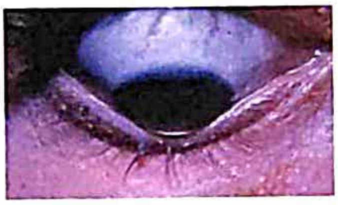
- Form of corneal extasia.
- Non-inflammatory, progressive, usuallly B/L.

Etiology :

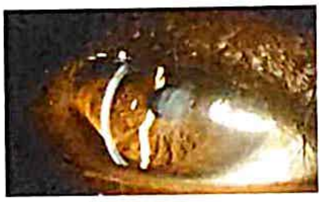
1. Congenital weakness.
2. Associated with :
    - Trauma.
    - Vernal Keratoconjunctivitis.
    - Down syndrome.

Clinical features :

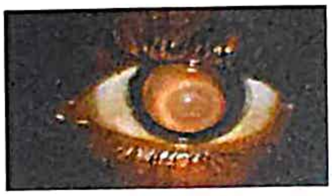
1. myopic astigmatism.
2. Hallmark : Stromal thinning (Central/ paracentral) → Inferior to the centre.
3. munson's sign : Bulging of lower eyelid on downgaze.
4. Rizzuti's sign : Light from temporal side → Arrowhead pattern of light over nasal limbus.
5. On retinoscopy : Scissoring reflex.
6. On ophthalmoscopy : Oil droplet reflex.
7. Vogt's striae : Stromal stress lines.



munson's sign



Rizzuti's sign



Oil droplet reflex

Diagnosis :

IOC: Corneal topography.

Treatment :

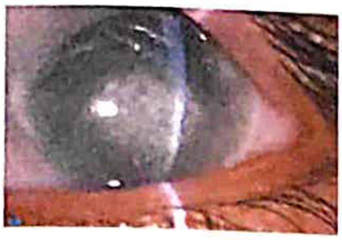
- Rigid contact lens (may lead to opacity).
- Keratoplasty.
- C3R (corneal collagen cross linking using riboflavin and UVA radiation).

Miscellaneous

00:27:54

Band shaped Keratopathy :

- D/T deposits of calcium in Bowman's layer.
- Treatment : Chelation with EDTA.

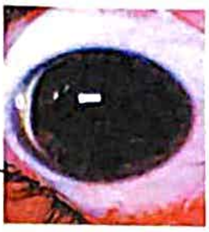


Band shaped Keratopathy

Arcus senilis :

- Occurs in old age.
- D/t lipid deposition on Descemet's membrane near periphery.

Lucid interval of Vogt : Clear zone of cornea b/w arcus and limbus.



Arcus senilis

Interstitial Keratitis :

Inflammation only of corneal stroma.

Causes :

- Congenital syphilis.
- Cogan syndrome (interstitial Keratitis + deafness).
- Tuberculosis.
- Herpes Simplex Virus (HSV).

Keratoplasty

00:32:57

Aka corneal transplantation.

TYPES

1. Penetrating Keratoplasty (Full thickness).
2. Lamellar Keratoplasty (Partial thickness)
    - DSEK (Descemet's stripping endothelial Keratoplasty).
    - DALK (Deep anterior lamellar Keratoplasty).

----- Active space -----

METABOLIC CAUSES OF DYSTROPHY

1. meesmann epithelial dystrophy.
2. macular dystrophy.
3. Schnyder central crystalline dystrophy.

TREATMENT OF CORNEAL DYSTROPHIES

If visual loss + : Keratoplasty/ Corneal transplantation.

00:15:54

Keratoconus

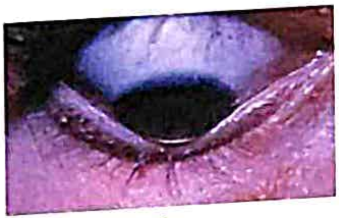
- Form of corneal extasia.
- Non-inflammatory, progressive, usuallly B/L.

Etiology :

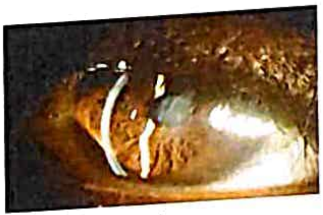
1. Congenital weakness.
2. Associated with :
    - Trauma.
    - Vernal Keratoconjunctivitis.
    - Down syndrome.

Clinical features :

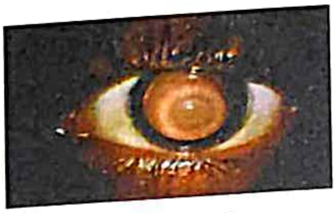
1. myopic astigmatism.
2. Hallmark : Stromal thinning (Central/ paracentral) → Inferior to the centre.
3. munson's sign : Bulging of lower eyelid on downgaze.
4. Rizzuti's sign : Light from temporal side → Arrowhead pattern of light over nasal limbus.
5. On retinoscopy : Scissoring reflex.
6. On ophthalmoscopy : Oil droplet reflex.
7. Vogt's striae : Stromal stress lines.



munson's sign



Rizzuti's sign



Oil droplet reflex

Diagnosis :

IOC: Corneal topography.

Treatment :

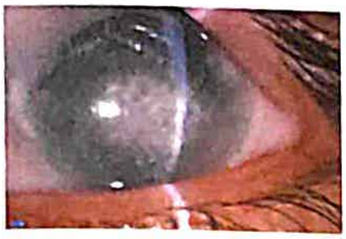
- Rigid contact lens (may lead to opacity).
- Keratoplasty.
- C3R (corneal collagen cross linking using riboflavin and UVA radiation).

Miscellaneous

00:27:54

Band shaped keratopathy :

- D/T deposits of calcium in Bowman's layer.
- Treatment : Chelation with EDTA.



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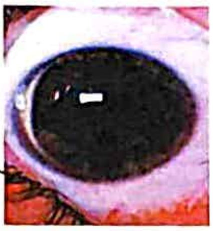

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00:32:57

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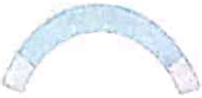
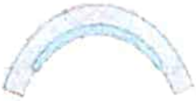
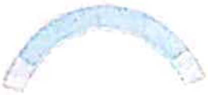
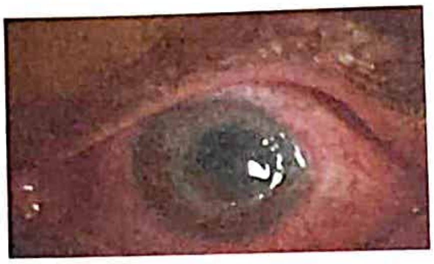
----- Active space -----

	Penetrating Keratoplasty	DSEK	DALK
Removal of	Epithelium to endothelium (i.e. all 5 layers)	Only endothelium & Descement's membrane (i.e. posterior 2 layers).	Corneal tissue from epithelium upto stroma (i.e. anterior 3 layers).
Indication	Pseudophakic bullous keratopathy.	-	Acute hydrops in severe keratoconus
			

Procedure :

Host cornea cut using a trephine → Implant the graft cornea.
(Diameter : 7.5mm) (Diameter : 0.25mm larger than host opening)

Trephine



Post-Keratoplasty

Graft rejection :

- Endothelial rejection (m/c).
  - Forms Khodadoust line (Diagnostic).

Storage media for corneal transplantation :

Type	Duration	Other features
Short term	<48hrs	4°C, moist chamber/McCarey Kaufman medium
Intermediate term	<2 weeks	-
Long term	upto 35 days	Cryopreservation (−70°C)

ANTERIOR UVEITIS

Classification of Uveitis

00:01:53

Anatomical Classification :

	Anterior Uveitis (AU)	Intermediate Uveitis	Posterior Uveitis	Pan Uveitis
AKA	Iridocyclitis.	Pars planitis/ Posterior cyclitis.	Chorioretinitis/ Retinochoroiditis	-
Involvement of	Iris : Iritis. Pars plicata : Cyclitis.	• Peripheral retina : Basal retinochoroiditis. • Vitreos : Vitritis/ Hyalitis.	Choroid & Retina	All layers of uvea.

Clinical Classification :

Acute :
Duration < 3 months.

Chronic :
Duration > 3 months +
Relapse in < 3 months

Recurrent :
≥ 3 months b/w 2 episodes

Etiological Classification :

Wood's classifica^n.

Granulomatous

Non-granulomatous

Clinical features of Anterior Uveitis

00:08:29

SYMPTOMS

- Pain : Referred along branches of 5th nerve.
- Redness
- Blurring of vision : D/t → Corneal haze
  → Aqueous turbidity
- Photophobia : Severe.
- Lacrimation.

SIGNS

1. Conjunctiva :

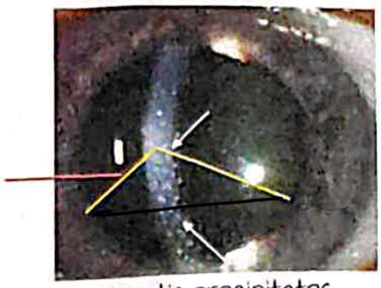
Circumcorneal congestion.

2. Cornea :

Corneal haze : D/t raised intraocular pressure (IOP)/ toxins.

Keratic Precipitates (KPs) :

- Cellular deposits on corneal endothelium.
- Location : Arlt's triangle (m/c).



Keratic precipitates

Type of KP	Seen in	Other features
Mutton fat	Granulomatous uveitis	Contain macrophages
Fine granules	Non-granulomatous uveitis	Contain lymphocytes
Pigmented	Chronic non-granulomatous uveitis	-
Stellate	Herpetic uveitis, Toxoplasmosis, Fuchs heterochromic iridocyclitis (FHI)	• Star-shaped • Diffuse distribution

Anterior Chamber :

1. Aqueous cells : markers of disease activity.

2. Aqueous flare :
   - Turbidity of aqueous : D/t leakage of proteins.
   - Visible d/t Tyndall effect/Brownian movement.

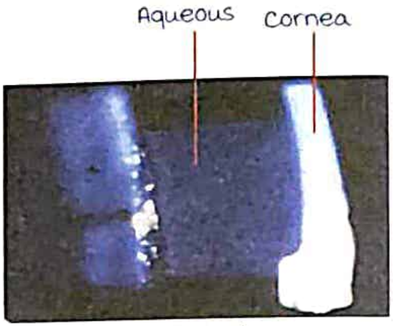
3. Hypopyon : WBCs pool infront of eye.

Hypopyon + Anterior uveitis : Behcet's disease.

4. Hyphema : Blood infront of eye chamber.

Hyphema + Anterior uveitis :

- Herpetic uveitis
- Toxoplasmosis
- Syphilis



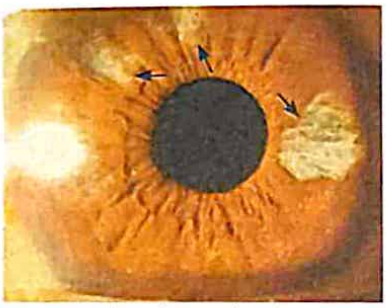
Aqueous cells

Iris :

1. Pseudo-rubiosis : D/t dilated iris vessels.

2. Muddy iris :
   - Seen in acute cases.
   - Aka water logged iris.

3. Iris atrophy :
   - Seen in chronic cases.
   - Types :
     - Sectoral : Herpetic uveitis
     - Diffuse : FHI

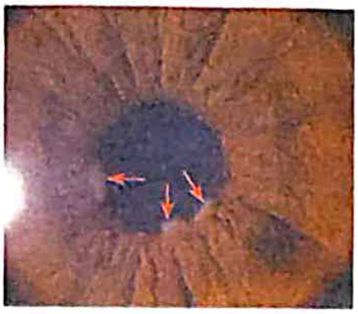


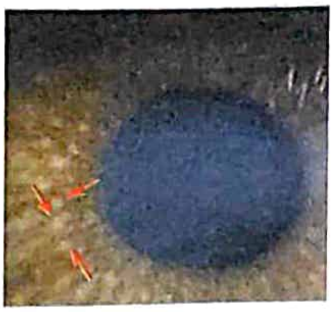
Sectoral iris atrophy

----- Active space -----

4. Iris nodules :

	Koeppe nodules	Busacca nodules:
Location	Pupillary margin	Iris stroma
Found in	• Granulomatous uveitis • Non granulomatous uveitis	Granulomatous uveitis


Koeppe nodules

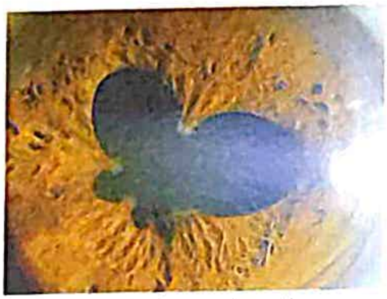

Busacca nodules

Pupil :

1. miosis : Acute cases.

2. Festooned pupil : Chronic cases.

Exudation → Formation of incomplete posterior synechiae
(Adhesion b/w pupillary margin and lens).
↓ Dilates the pupil
Irregular dilation.

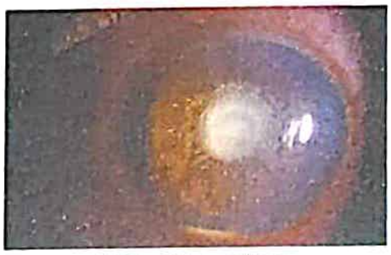

Festooned pupil

3. Fixed pupil :
- Recurrent cases.
- Annular/Ring/Complete posterior synechiae (360° adhesion) → No aqueous flow from posterior chamber to anterior chamber → Secondary angle closure glaucoma.

4. Total Posterior Synechiae (TPS) :
Exudation in posterior chamber → Adhesion b/w posterior surface of iris and lens.

5. Cyclitic membrane : TPS + Exudates leak behind lens.
Covers ciliary processes, equator & posterior surface of lens → No aqueous secretion → ↓ IOP.

6. Occlusio pupillae : Exudates organised across anterior surface of pupil.


Occlusio pupillae

IOP :

Acute AU :

↑ IOP : D/t trabecular meshwork blockage by cells and proteins + Associated trabeculitis.

Chronic AU :

↓ IOP : D/t cyclitic membrane + Shut down of ciliary body

Complications and Treatment of Anterior Uveitis

00:45:26

Complications :

1. Complicated Cataract : m/c
Bread crumb appearance
+ polychromatic lustre.

2. Secondary angle closure glaucoma :
m/c in recurrent cases.

3. Band shaped keratopathy :
Calcium deposits in Bowman's layer of cornea.

Note :

Causes of complicated cataract :

Mnemonic : UMAR

- Uveitis.
- Myopia.
- Angle closure glaucoma.
- Retinitis pigmentosa.

4. Cystoid macular edema :
M/C cause of visual loss d/t Posterior involvement in anterior uveitis (AU).

Treatment :

Topical steroids :

- Prednisolone.
- Difluprednate.

Antimetabolites : Azathioprine.

- Given if inflammation + recurrent episodes requiring long term steroids.

Cyclopegics : Atropine

Action :

- Relax ciliary muscle → Pain relief.
- Break posterior synechiae.
- Prevent formation of posterior synechiae.

Note :

Side effects of steroids :

- Topical administration : Glaucoma.
- Systemic administration : Cataract.

Causes of Anterior Uveitis

00:52:39

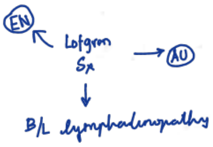
GRANULOMATOUS

a. Tuberculosis.

b. Leprosy :

- m/c : Leprosy Lepromatous leprosy.
- Iris pearls : Pathognomic feature.

Iris Pearls.



c. Sarcoidosis :

- m/c ocular manifestation of sarcoidosis : Granulomatous anterior uveitis.
- Lofgren syndrome: Erythema nodosum + B/L lymphadenopathy + Anterior uveitis.
- Heerfordt syndrome/Uveoparotid fever.

d. Syphilis :

- m/c in late congenital syphilis.
- Presentation : Iris roseolas.

Iris Roseolas .

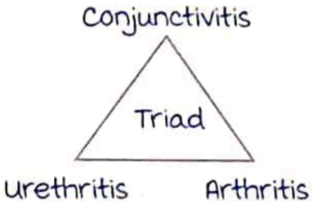
e. Herpes :

AU + Raised IOP + Hyphema + Stellate KPs + Sectoral iris atrophy.

2. NON GRANULOMATOUS

Acute :

1. Idiopathic : m/c
2. HLA B27 associated disease :
   - Ankylosing Spondylitis : Lower back pain + stiffness + recurrent U/L AU.
   - Reiter's disease :



+ U/L AU + Keratoderma blenorrhagicum + Circinate balanitis + History of travel.

- Psoriatic arthritis : Sausage digits + B/L AU.

3. Behcet's disease :

- A/W HLA B5, B51.
- B/L recurrent AU + Cold mobile hypopyon + Aphthous mouth ulcers.

4. Inflammatory bowel disease :

Ulcerative colitis (12%) > Crohn's disease (2.4%).

5. Drug induced uveitis :

----- Active space -----

Mnemonic : CML Please Relapse Slowly.

- Cidofovir.
- Metipranolol.
- Latanoprost.
- Pilocarpine.
- Rifabutin.
- Sulfonamides.

Chronic :

Juvenile Idiopathic Arthritis (JIA) :

- M/C cause of AU in paediatric age group.
- Types :
  - Pauciarticular : ≤4 joints involved
    - Type 1 :
      - M/C in Females.
      - M/C A/W AU.
      - RF -ve (Seronegative).
      - ANA +ve.
    - Type 2 :
      - M/C in males.
  - Polyarticular : >4 joints involved
  - Stills disease

2. Fuch's Heterochromic Iridocyclitis (FHC) :

- Clinical features :

Mnemonic : No FUCHS.

- No posterior synechiae.
- Floaters.
- Unilateral.
- Cataract.
- Heterochromia iridis : Hypochromia in the affected eye d/t loss of iris crypts.
- Stellate Keratic precipitates.

- Amsler's Sign : Bleeding on paracentesis.

INTERMEDIATE, POSTERIOR, PAN-UVEITIS AND MISCELLANEOUS DISORDERS

Intermediate Uveitis

00:00:07

Also known as pars planitis.

Causes :

1. Idiopathic (m/c).
2. Associated with:
   - Lyme's disease.
   - Sarcoidososis.
   - Multiple sclerosis.
   - Tuberculosis.

Clinical features :

Symptoms :

- B/L floaters.
- Blurring of vision.

Signs :

- Spillover anterior uveitis.
- Snowballs : Aggregation of cells in vitreous.
- Snowbanking : Fibrovascular plaque.
- Cystoid macular edema : m/c cause of vision loss.

Treatment :

Steroids : Posterior subtenon injection

↓ If fails

Steroids : Systemic

↓ If fails

Immunomodulatory drugs : Azathioprine

↓ If fails

Pars plana vitrectomy + Photocoagulation of snowbank

Posterior Uveitis/Chorioretinitis

00:09:17

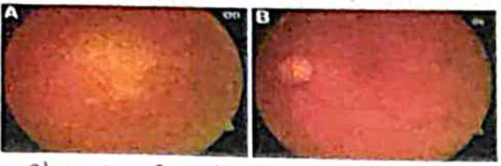
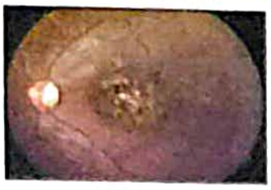
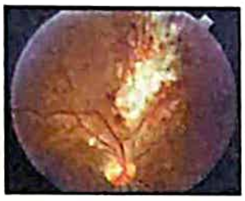
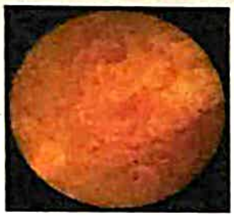
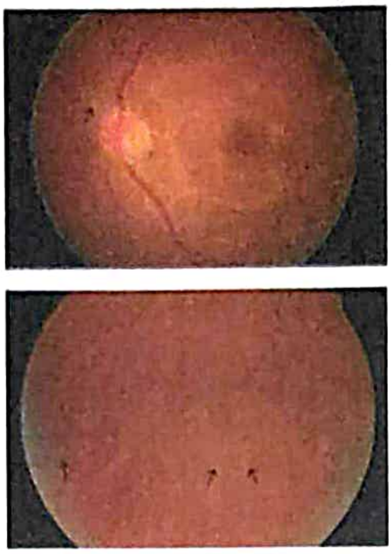
Inflammation of choroid & retina.

Clinical features :

1. Floaters.
2. Painless loss of vision.
3. Scotoma.
4. metamorphopsia : Distortion of image.
5. Photopsia.

----- Active space -----

Causes :

Infectious	Non-infectious
1. most common : Toxoplasmosis. a. Active : Headlight in fog appearance (d/t associated active viritis.)  b. Chronic : Punched out scar.  - Cats are definitive hosts. - Toxoplasmosis causes focal chorioretinitis. 2. Toxocariasis : - Unilateral LOV. - Leukocoria (white pupillary reflex). 3. CMV retinitis : - m/c cause of LOV in ocular HIV. a. Pizza pie appearance. b. Scrambled egg & ketchup appearance.  c. Brushfire extension.  4. TB	1. Sarcoidosis : - Candle wax dripping appearance. (D/t perivascular inflammation & sheathing).  - Lander's sign : Pre-retinal nodules. 2. Behcet's disease. 3. Birdshot chorioretinopathy : HLA A29 association. 4. Serpiginous choroiditis : HLA B7 association.

Focal chorioretinitis : Toxoplasma, Toxocariasis, CMV.

Multifocal chorioretinitis : HSV, TB, syphilis.

Treatment :

----- Active space -----

1. Infectious cases : Treat the cause + systemic steroids.
2. Non-infectious cases : Systemic steroids

OR

- Adalimumab (visual studies I to II)
- Sirolimus (Sakura program).
- Voclosporin (Luminate trials).

Pan-uveitis

00:22:27

Causes :

1. Vogt-Koyanagi-Harada (VKH) syndrome :

Affects :

Eye
- B/L granulomatous panuveitis.
- Sunset glow fundus : RPE atrophy.
- Suiguira sign : Perilimbal vitiligo.
- Exudative RD.

Ears : Tinnitus

Skin : Vitiligo

Meninges : Meningoencephalitis

Poliosis → Loss of lashes & regrowth of depigmented while lashes.

2. Sympathetic ophthalmitis :

- Clinical feature : Granulomatous panuveitis.
- Cause : Accidental penetrating trauma affecting ciliary body.
- Pathogenesis : Trauma to one eye / Exciting eye

↓ >2 weeks

Sympathetic ophthalmitis in other eye (sympathizing eye).

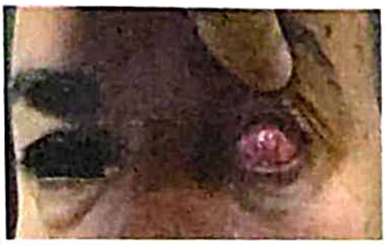
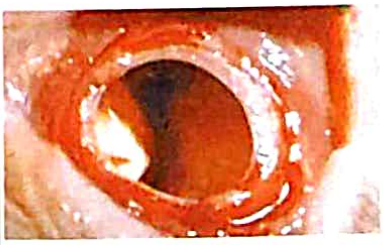
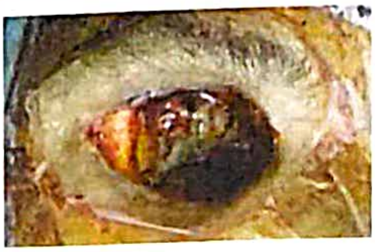
Prevention : Enucleation of traumatic eye in 14 days.

Treatment : Steroids.

----- Active space -----

Miscellaneous

00:33:00

	Enucleation	Evisceration	Exentration
Terminology	Removal of whole eyeball + optic nerve	Removal of all layers of eyeball except sclera.	Removal of eye and orbital contents.
Indications	• Retinoblastoma. • Trauma.	Endophthalmitis.	• Orbital mucormycosis. • Tumors metastasised to orbit.
C/I	Panophthalmitis	Retinoblastoma	-
Appearance			

Gyrate atrophy :

- Type of choroid atrophy.
- Autosomal recessive.
- mutation in ornithine aminotransferase gene : ↑Levels of ornithine.
- Treatment : Arginine restriction in diet (ornithine is synthesised from arginine)

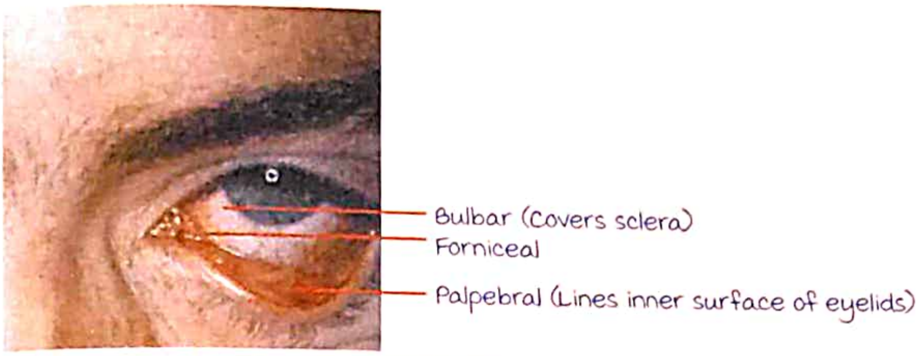
ANATOMY OF CONJUNCTIVA, TYPES OF CONJUNCTIVITIS AND PTERYGIUM

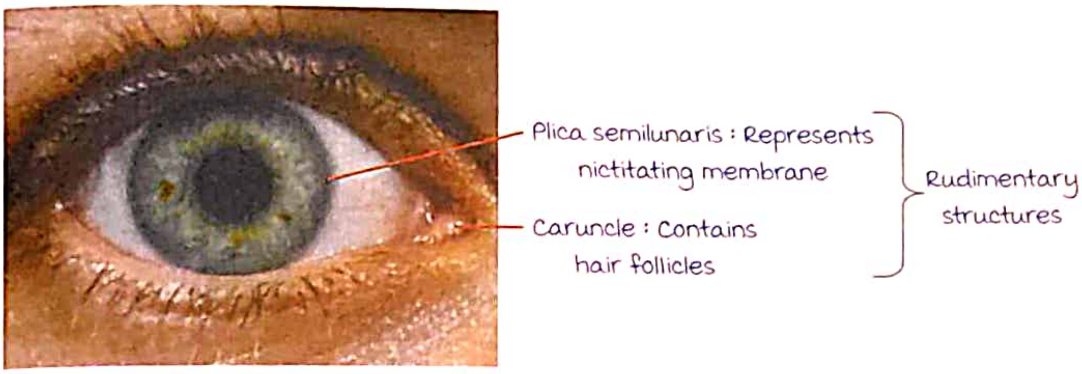
Conjunctiva is transparent mucous membrane.
It does not cover cornea.

Anatomy

00:00:50

Parts of conjunctiva :


Bulbar (Covers sclera)
Forniceal
Palpebral (Lines inner surface of eyelids)


Plica semilunaris : Represents nictitating membrane
Caruncle : Contains hair follicles
Rudimentary structures

Histology :

1. Outermost layer : Epithelium (Non-Keratinised stratified squamous).

Contains :

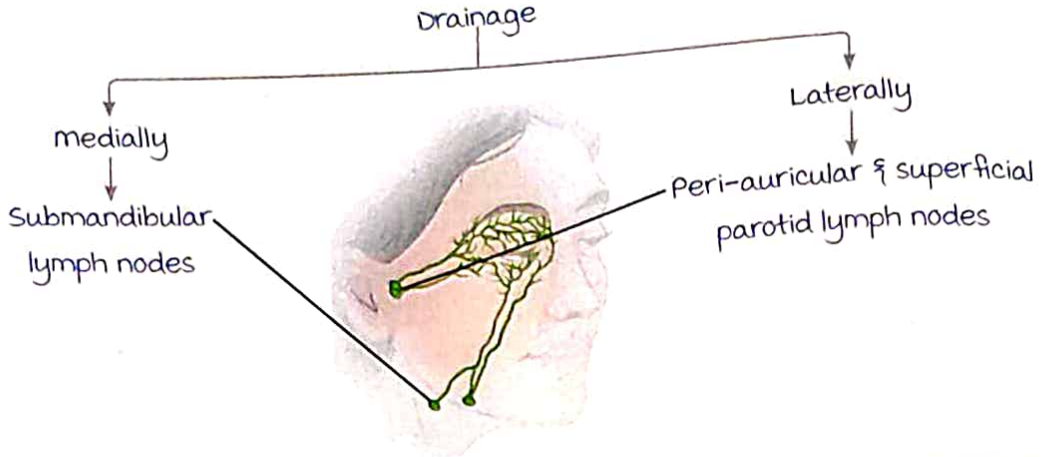
- a. Goblet cells : Secrete mucin (Forms innermost layer of tear film) on parasympathetic stimulation.
- b. Melanocytes.
- c. Langerhans cells.

2. Adenoid/lymphatic layer : Develops after 3 months of age.

3. Fibrous layer : Contains nerve fibres & blood vessels.

Lymphatic drainage :

Only site of lymphatic drainage from the eye.


Drainage
Medially
Submandibular lymph nodes
Laterally
Peri-auricular & superficial parotid lymph nodes

00:08:40

Conjunctivitis

Aka Eye flu.

Symptoms :

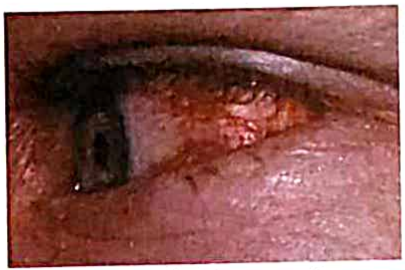
- Redness.
- Discharge sticking to eyelids.
- Foreign body sensation.
- Lacrimation.
- Itching (If allergic).
- Pain and loss of vision (LoV) : Corneal involvement.

Signs :

1. Conjunctival hyperemia (Redness) :

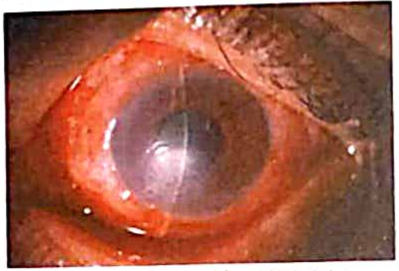
Differential diagnosis :

Circumcorneal redness seen in → Glaucoma
→ Uveitis
→ Corneal ulcer



Conjunctival hyperemia

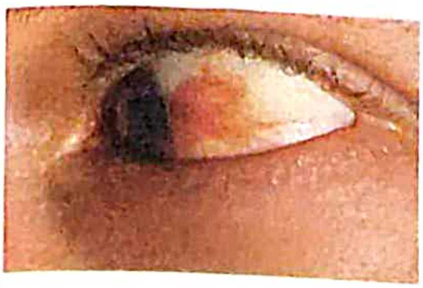
Note :



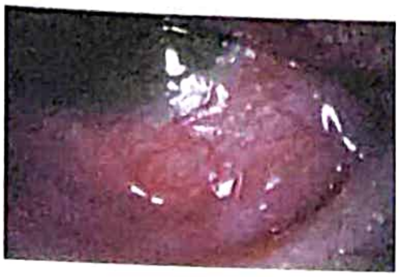
Circumcorneal hyperemia

- Seen in : Glaucoma, uveitis, corneal ulcer.

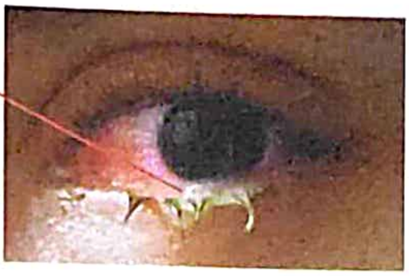
2. Subconjunctival hemorrhage :
No treatment required (Resolves spontaneously ≤ 2 weeks).



3. Chemosis : Swelling of bulbar conjunctiva.

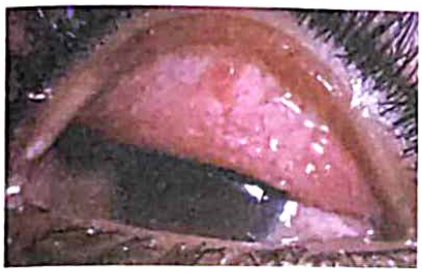


4. Discharge : mucopurulent/watery.
- Causes colored halos & stickiness which resolves on washing eye.

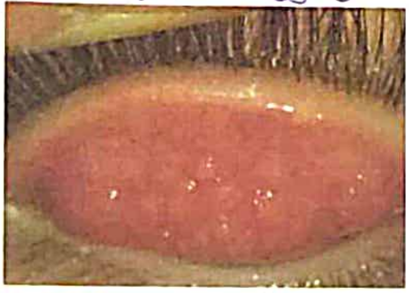


5. Inflammatory reactions of conjunctiva :

Papillae : Hypertrophied vessels.



Follicles : Lymphoid aggregates.



Etiology :

Discharge	Papillary reaction	Follicular reaction
Mucopurulent	Bacterial	Chlamydia
Watery	Allergic	Viral

Bacterial conjunctivitis

00:17:22

Acute mucopurulent conjunctivitis :

M/C cause : Staphylococcus aureus.

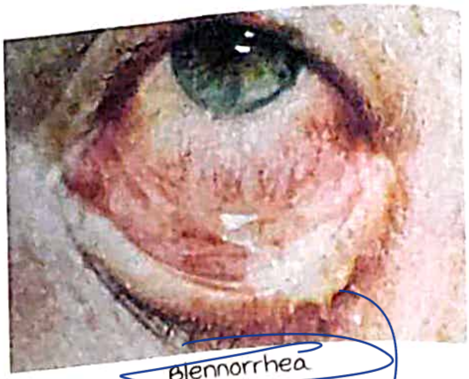
Treatment : Topical antibiotics.

----- Active space -----

Acute purulent/hyperacute conjunctivitis/Blenorrhoea :

m/c cause : Neisseria gonorrhoeae.

Route of transmission : Oculogenital.



Clinical features :

1. Copious purulent discharge.
2. Swelling of eyelids → Overhanging.
3. Intense pain.
4. Pre-auricular lymphadenopathy.

Treatment :

I.m. Ceftriaxone 1g single dose
↓ followed by
Oral erythromycin x 2 weeks.

Acute membranous conjunctivitis :

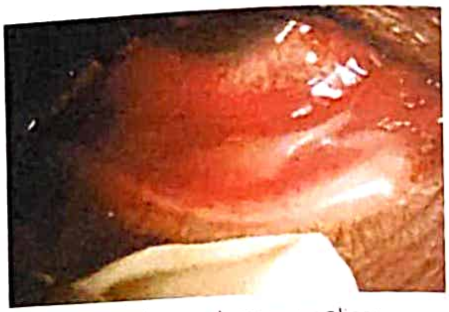
m/c cause
→ Overall/in adults : Pneumococcus.
→ In unimmunised children : Corynebacterium diphtheriae.

Clinical features :

membrane formed → Fused with epithelium → Bleeds on removal.

Acute pseudomembranous conjunctivitis :

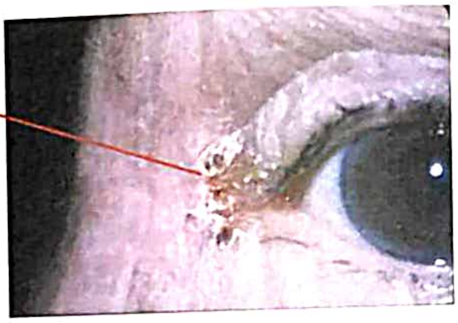
membrane does not fuse with epithelium
↓
No bleeding on removal.



Pseudomembrane peeling

Angular/diplobacillary conjunctivitis :

m/c cause : moraxella lacunata/axenfeld



Clinical features :

- Excoriation at canthi (Lateral > medial).
- Redness at intermarginal strip.
- Blepharitis (Inflammation of eyelid margin).

Treatment :

- Tetracycline 1% eye ointment for ≥ 2 wks.
- Zinc boric eye drops. (Zinc blocks proteolytic activity).

Viral Conjunctivitis

00:27:00

----- Active space -----

Adenoviral conjunctivitis :

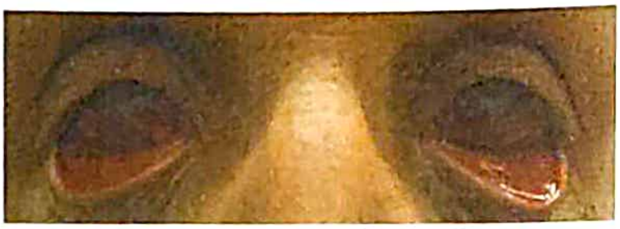
Non-specific follicular conjunctivitis :
Serovars : 1 to 11 & 19.

Epidemic Keratoconjunctivitis (EKC) :
Serovars : 8, 19, 37

Pharyngoconjunctival fever (PCF) :
- Serovars : 3, 4, 7.
- Associated with preauricular lymphadenopathy

Apollo conjunctivitis/Acute hemorrhagic conjunctivitis :

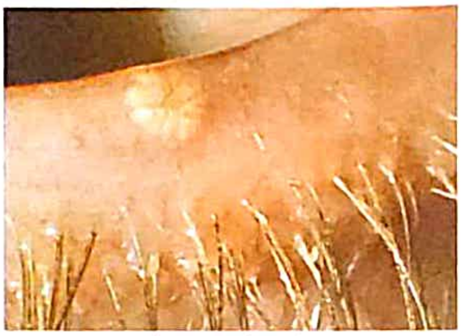
Hemorrhage of palpebral & bulbar conjunctiva.



Causes :

1. Picornavirus : Enterovirus type 70, Coxsackie A24 (m/c).
2. Adenovirus type 11.

molluscum contagiosum :



Umbilicated nodule

Clinical features :

- Unilateral, painless nodule with umbilicated appearance.
- Nodule contains viral particles.

↓

Periodic shedding of virus

↓

Conjunctivitis

Treatment : Surgical excision [If cosmetic indication (+)].

Chlamydial Conjunctivitis

00:33:14

Adult inclusion conjunctivitis :
D to K serovars

Trachoma :
A, B, Ba, C serovars

Ophthalmia neonatorum :
- D to K serovars
- Other causes

TRACHOMA

Aka Egyptian ophthalmia.

Route of transmission : mnemonic : 3F.

1. Fingers.
2. Flies.
3. Fomites.

----- Active space -----

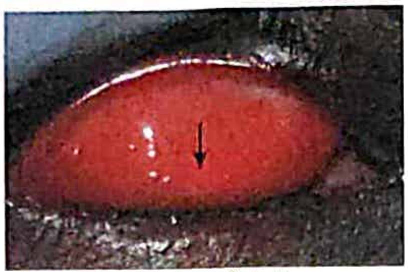
Pathology :

Type IV hypersensitivity reaction : Active inflammation + cicatrization.

Age group : < 10 yrs.

Clinical signs :

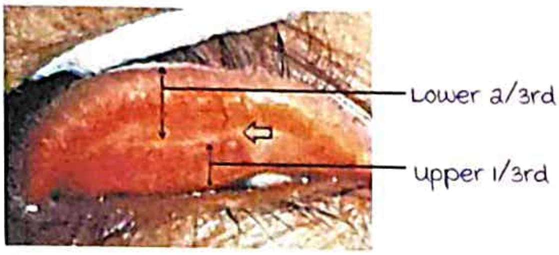
1. Sago grain follicles : Necrosis & leber.
   - m/c site : upper palpebral conjunctiva.
   - Necrosis & leber cells seen.



Sago grain follicles

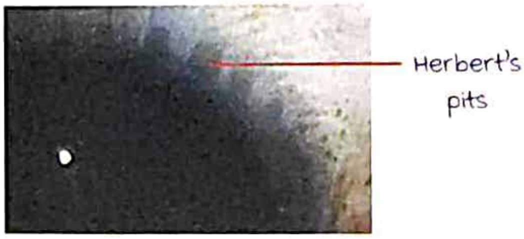
2. Cicatricial sign :

a. Arlt's line : Line of cicatrization.

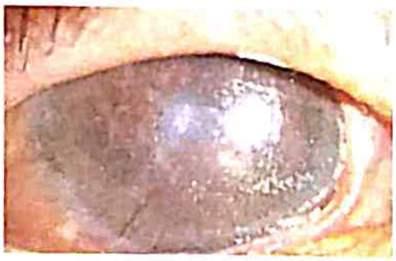


b. Herbert's pits : Pathognomic.

- D/t healing of limbal follicles.



c. Pannus : Vascularisation of cornea superiorly.



Complications :

Trichiasis (Inturned eyelash) → Corneal ulcer → Scar formation → Loss of vision.

Treatment :

SAFE strategy (WHO, 1996) :

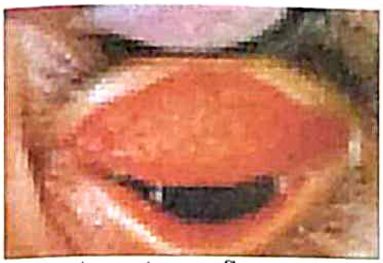
- Surgery for inturned eyelids.
- Antibiotics : Azithromycin 1g.
- Facial cleanliness.
- Environmental change.

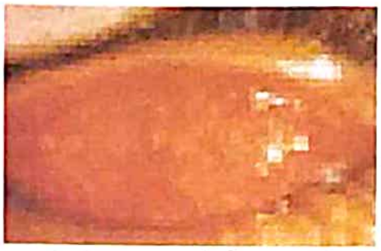
Indications for treatment :

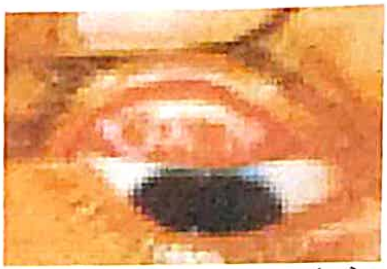
Prevalance in 1-9 yr old children	management
> 10 %	mass prophylaxis
5-10 %	Treatment to affected children & family
< 5 %	Facial cleanliness & environmental change advised

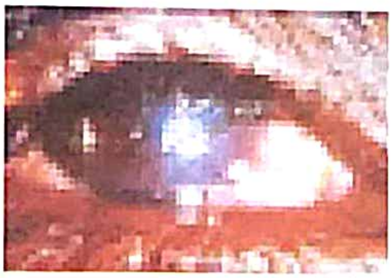
FISTO CLASSIFICATION (WHO)

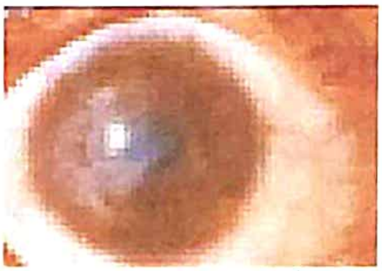
	Features	management
TF : Trachomatous inflammation follicular	> 5 follicles +	Active stage Treatment : Antibiotics
TI : Trachomatous inflammation intense	Thickening of upper palpebral conjunctiva	
TS : Trachomatous scarring	Arlt's line	Inactive stage : No treatment
TT : Trachomatous trichiasis	Eyelash growing inwards	Requires surgery
CO : Corneal opacity	Visual impairment	-


Trachomatous inflammation follicular (TF)


Trachomatous inflammation follicular and intense (TF)

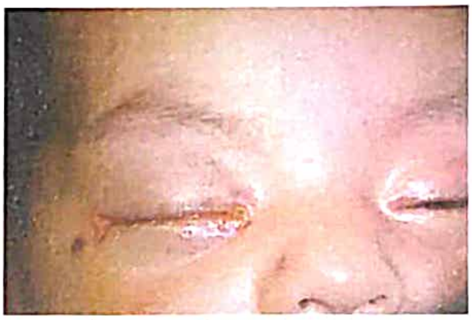

Trachomatous scarring (TS)


Trachomatous trichiasis (TT)


Corneal opacity (CO)

OPHTHALMIA NEONATORUM

Conjunctivitis in neonates (< 28 days of onset).


Ophthalmia neonatorum

Causes :

Onset (Hours after birth)	Cause
Within first 6h	Chemical conjunctivitis (Silver nitrate)
24 to 48h	Neisseria gonorrhoeae (most severe)
2 to 5 d	Other bacteria
5 to 7 d	HSV-II
> 1 wk	Chlamydia trachomatis (D to K) : m/c

----- Active space -----

Prevention :

1. Crede's method : Topical 1% Silver nitrate (Not recommended d/t chemical conjunctivitis).
2. 0.5% erythromycin. } • Single application.
3. 1% tetracycline. } • Within 1 hr of birth.

Allergic conjunctivitis

00:50:24

Presents with papillae reaction + watery discharge + itching.

VERNAL KERATOCONJUNCTIVITIS/SPRING CATARRH

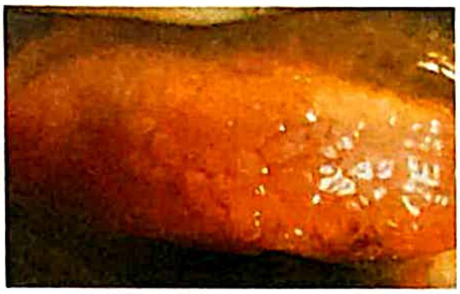
Type I hypersensitivity reaction (Eg : pollen).

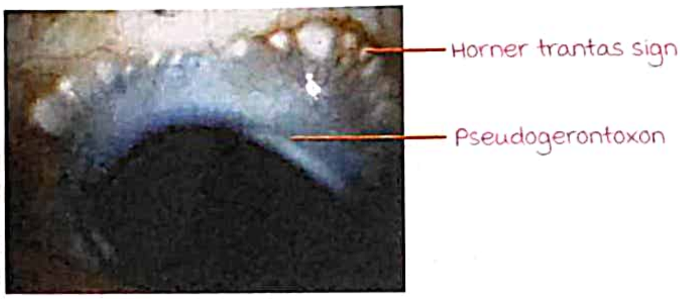
Incidence :

- Age : 5-15 yrs.
- Males > Females (Young boys).
- M/C in spring & summer.
- H/O atopy present.

Clinical signs :

1. Papillary hypertrophy :
   - Cobble stone papillae.
   - Giant papillae : > 1mm size.
2. Horner Trantas sign :
   - Collection of eosinophils + epithelial debris.
   - Site : On limbus.





3. Pseudogerontoxon : Paralimbal grey-white band (D/t lipid deposition) in children.
   - Appearance similar to arcus senilis.
4. Shield ulcer (In cornea).
5. Dennie morgan line : Extra lower-lid crease.
6. Maxwell Lyon sign : Ropy discharge.

Note : Excessive itching
↓
↑ incidence of Keratoconus.

Treatment :

1. Cold compress.
2. Topical anti-histamine.
3. Topical mast cell stabilisers.
4. DOC : Olopatadine or Alcaftadine.
5. Topical steroids in acute exacerbation (For symptomatic treatment).

ATOPIC KERATOCONJUNCTIVITIS

Seen in 2nd to 5th decade.

Clinical features :

- Eyelids : Hertoghe sign (Lateral eyebrow lost).
- Itching.
- Papillae.
- Watery discharge.

Treatment :

1. Topical anti-histamine.
2. Topical mast cell stabilisers.

GIANT PAPILLARY KERATOCONJUNCTIVITIS

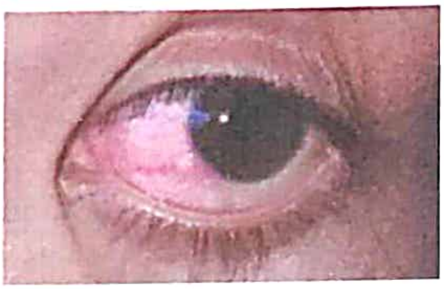
- Papillae > 1mm.
- Type IV hypersensitivity reaction.

Etiology : mechanically induced by
→ Contact lens
→ Ocular prostheses
→ Protruding sutures

Treatment : Anti-histamine + surgery to remove irritant (If required).

PHLYCTENULAR KERATOCONJUNCTIVITIS

Type IV hypersensitivity reaction in response to endogenous allergen.


Phlycten

Etiology :

- m/c in India : TB.
- m/c in western countries : Staphylococcus aureus.

Clinical features :

- Phlycten : Nodule near limbus.
  - Grows towards cornea → Ulcerates.
    Gives rise to
    → Sacrofulous ulcer
    → Fascicular ulcer
    → Miliary ulcer

Treatment : Topical Steroids.

----- Active space -----

Pterygium

01:07:20

Triangular, fibrovascular growth of degenerative sub-conjunctival tissue over Cornea.

- Destroys Bowman's layer & superficial stroma.
- Associated with → UV ray exposure
  → Dust
  → Humidity

Cause : Limbal stem cell deficiency → Activation of matrix metalloproteinase.

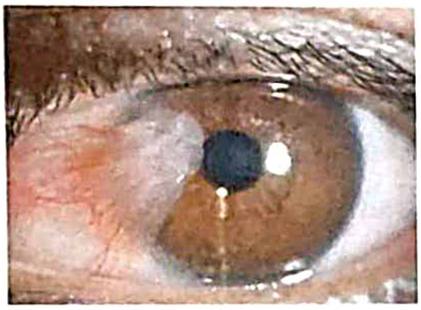
Site : Nasal > Lateral.

Clinical features :

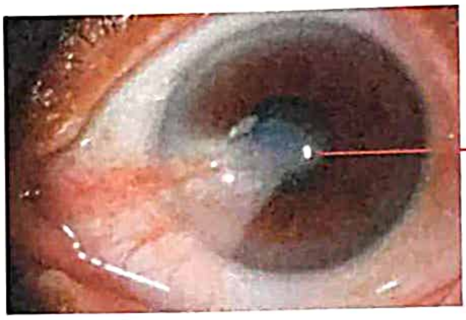
1. Triangular growth with apex towards cornea.
2. Stocker's line : Deposition of iron in front of apex.
3. Loss of vision.

Causes :

a. Corneal astigmatism.



b. Encroaching into visual axis.



Covers pupillary area

Treatment :

Surgical excision : ↑ rate of recurrence.

- To reduce recurrence :

1. Topical mitomycin C.
2. Autograft : Harvested from same eye.
   - Site : Usually superior.
   - Fixed with sutures/fibrin glue/autologous serum.

Pinguecula & concretions

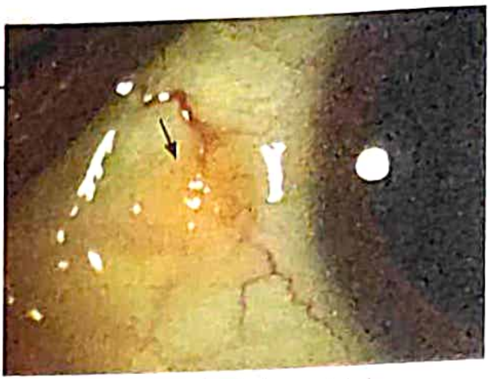
01:14:28

----- Active space -----

PINGUECULA

Elastic degeneration of collagen fibres in stroma of conjunctiva.

m/c site : Nasal part of conjunctiva.



Yellowish fat-like nodules

CONCRETIONS

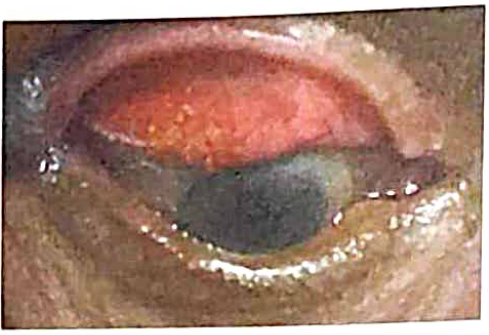
Collection of epithelial debris & mucus.

Cause : Irritation d/t friction of eyelid against bulbar conjunctiva & cornea while blinking.

Treatment : Removal with 26g needle.

- Spontaneously resolves with good lubrication.

Note : Concretions do not contain calcium deposits.



minute yellowish-white elevations

----- Active space -----

EYELIDS : ANATOMY AND PATHOLOGIES

00:01:01

Anatomy

3 muscles :

Muscle	Nerve	Function	Presentation in palsy
Levator palpebrae superioris	CN III	Elevation of upper lid	Ptosis
Müller's muscle	Sympathetic fibres		
Orbicularis oculi	CN VII	Closure of eyelid	Lagophthalmos

3 glands :

Gland	Type of gland	Opens at
Meibomian (tarsal)	Modified sebaceous	Posterior to anterior surface of eyelid margin
Zeis		Base of lash follicles
Moll	Modified sweat	

EYELID

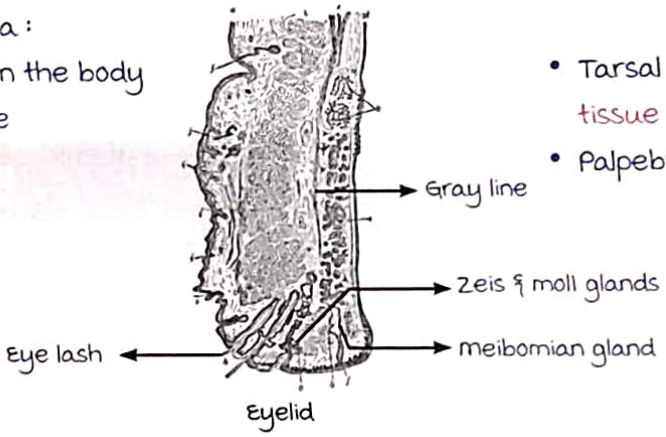
Grey line : Divides eyelid into

Anterior lamina :

- Skin : Thinnest skin in the body
- Subcutaneous tissue
- Muscles

Posterior :

- Tarsal plate has only fibrous tissue
- Palpebral conjunctiva

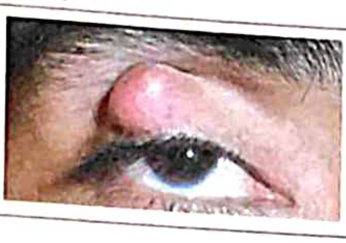
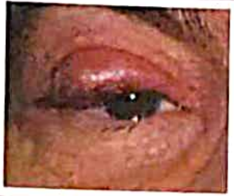


Pathologies

00:09:16

CHALAZION & HORDEOLUM

	Chalazion	External hordeolum / stye
Pathology	Lipogranulomatous inflammation of meibomian gland	Suppurative inflammation of Zeis gland
Infectivity	Non-infective	Infective : S. aureus

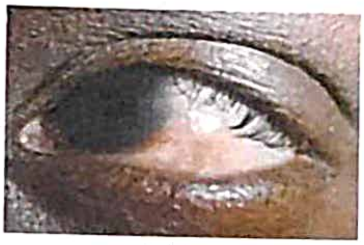
	Chalazion	External hordeolum / stye
Swelling	• Painless • Circumscribed swelling away from lid margin	• Painful • Diffuse swelling at the lid margin.
Rx	• Incision & curettage : – Horizontal incision on posterior part of eyelid (conjunctiva). • Intralesional triamcinolone	• Hot compresses • Oral antibiotics if recurrent.
Complications	Recurrence → Sebaceous cell carcinoma	–
Appearance		

Hordeolum internum :

Suppurative inflammation of meibomian gland

TRICHIASIS

- Inward misdirection of eyelashes
- Causes corneal opacity

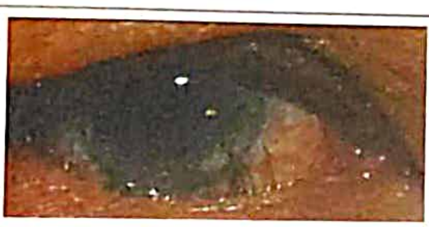
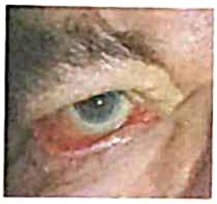


Trichiasis

Treatment

- Epilation every 1–2 months.
- Cryotherapy of lash base : Definitive.

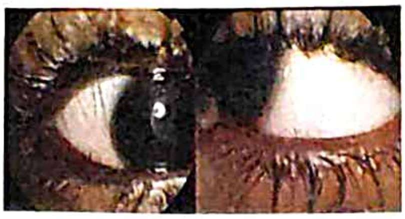
ENTROPION & ECTROPION

	Entropion	Ectronion
Feature	• Inward turning of eyelid margin	• Outward turning of eyelid margin m/c cause : Senility
Rx :	 • Spastic → Botulinum toxin injection • Senile → modified wheeler operation, Weiss Operation • Cicatricial → modified Burrows operation, Wedge resection	 • medial conjunctivoplasty • Buryon Smith operation

----- Active space -----

DISTICHIASIS

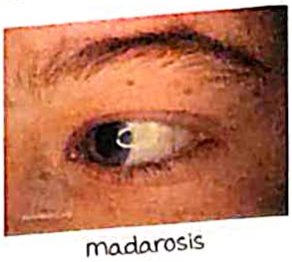
Extra posterior row of eye lashes.



Distichiasis

MADAROSIS

Loss of lateral 1/3rd of eye lashes & eyebrow d/t chronic rubbing of the eye.



madarosis

TYLOSIS

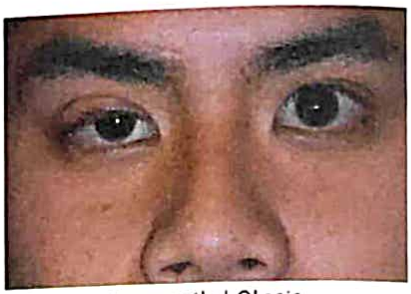
Thickening of eyelid margin.

ANKYLOBLEPHARON

- Fusion of eyelid margin of upper & lower lid.
- Post trauma.

CONGENITAL PTOSIS

D/t defective development of levator palpebrae superioris.



Congenital Ptosis

Clinical features :

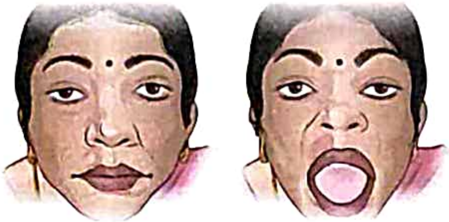
- Absent upper lid crease.
- Lid lag on downgaze :
  - Failure of eyelid to go down on downward gaze.

Treatment :

Ptosis	LPS function	Treatment
moderate	Fair	LPS resection
Severe	Poor	Frontalis sling SX

Marcus Gunn Jaw winking syndrome :

D/t trigemino - oculomotor nerve synkinesis.



Closed jaw → ptosis Open jaw → lid moves up

ANATOMY OF ORBIT AND PROPTOSIS

Bony cage surrounding eye.

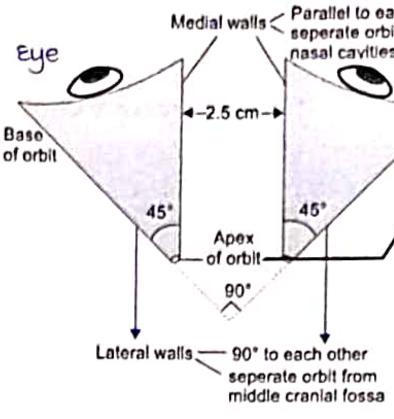
Anatomy of Orbit

00:00:31

WALLS OF ORBIT

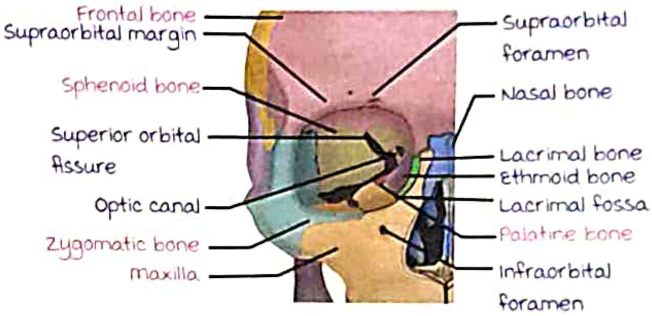
Wall	Constituent bones
Roof	• Frontal bone. • Lesser wing of sphenoid.
Lateral wall	• Zygomatic bone • Greater wing of sphenoid
Floor (m/c to fracture → Blow out fracture)	• maxilla. • Zygomatic bone. • Palatine bone
medial wall → Thinnest wall (Lamina papyracea)	Mnemonic: SMEL • Sphenoid : Body. • maxilla : Frontal process. • Ethmoid bone. • Lacrimal bone.

Note: Eyeball naturally protrudes slightly from lateral wall of orbit.


Medial walls
Parallel to each other
seperate orbit from
nasal cavities
Eye
Base
of orbit
2.5 cm
45°
45°
Apex
of orbit
90°
Lateral walls
90° to each other
seperate orbit from
middle cranial fossa

Contains Annulus of Zinn → Origin of all extra-ocular muscles (except Inferior Oblique)

Orbit : Schematic


Frontal bone
Supraorbital margin
Sphenoid bone
Superior orbital fissure
Optic canal
Zygomatic bone
Maxilla
Supraorbital foramen
Nasal bone
Lacrimal bone
Ethmoid bone
Lacrimal fossa
Palatine bone
Infraorbital foramen

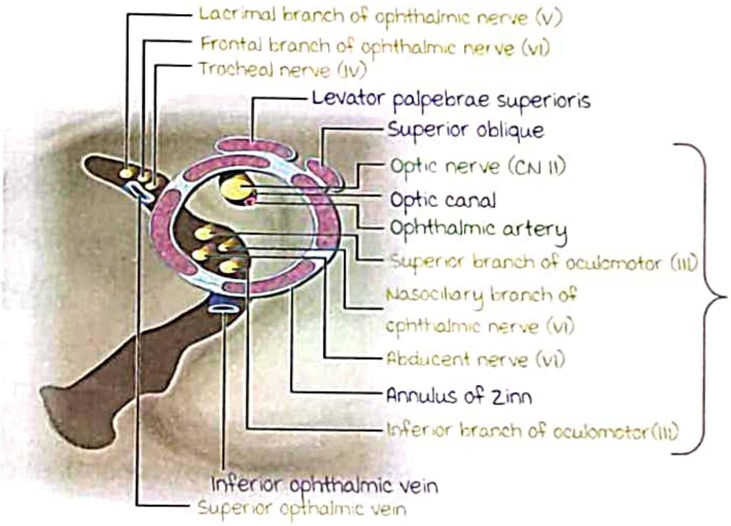
Bones forming orbit

OPENINGS INTO ORBITAL APEX

a. Optic canal.

b. Superior orbital fissure.
(Between roof and lateral wall)

c. Inferior orbital fissure.
(Between floor and lateral wall)


Lacrimal branch of ophthalmic nerve (V)
Frontal branch of ophthalmic nerve (VI)
Trocheal nerve (IV)
Levator palpebrae superioris
Superior oblique
Optic nerve (CN II)
Optic canal
Ophthalmic artery
Superior branch of oculomotor (III)
Nasociliary branch of ophthalmic nerve (VI)
Abducent nerve (VI)
Annulus of Zinn
Inferior branch of oculomotor (III)
Inferior ophthalmic vein
Superior opthalmic vein

Structures passing through the Annulus of Zinn

Structures passing through the openings

----- Active space -----

Proptosis/Exophthalmos

00:13:17

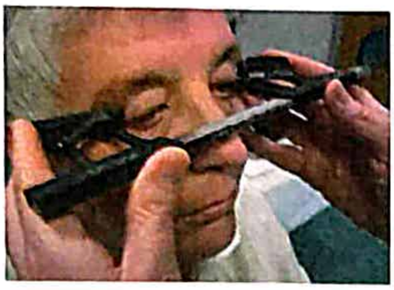
Protrusion of eyeball from lateral orbital rim :

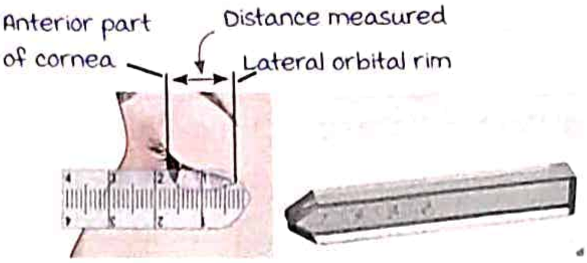
- >21mm. OR
- 2mm difference between 2 eyes.

MEASUREMENT

Exophthalmometer

- **Hertel's**
  - Can measure both eyes together.
  - Preferred overall.
- **Leude's**
  - Only measures 1 eye at a time.
  - Preferred in children.





CAUSES

Bilateral :

- **Painless :**
  - Grave's disease.
  - Cortico-cavernous fistula.
  - Leukemia/ AML (in children)
- **Painful :**
  - Cavernous sinus thrombosis (CST).
  - Grave's (restriction of ocular movement causing pain)

Unilateral :

- **Axial :**
  - Along 1° axis.
  - Outward, straight protrusion.
  - **Painful :**
    - Orbital cellulitis.
    - CST
  - **Painless :**
    - ON glioma
    - Cavernous hemangioma
- **Abaxial :**
  - Away from 1° axis.
  - Direction depends on cause.
  - **Inferomedial :**
    - Superotemporal lesions.
    - Lacrimal gland tumors.
    - Dermoid cysts.
  - **Upward :** maxillary sinus tumor
  - **Lateral :** Lacrimal sac

m/C causes :

In adults : Grave's disease. (Unilateral or bilateral proptosis.)

In children :

a. U/L : Orbital cellulitis.
b. B/L : Leukemia/AML.

Inflammatory Causes of Proptosis

00:23:23

CAVERNOUS SINUS THROMBOSIS & ORBITAL APEX SYNDROME

	Cavernous sinus thrombosis	Orbital apex syndrome
Structures involved (structures passing through)	CN III, IV, VI, V_1, V_a	• Contains structures passing through superiod orbital fissure & optic canal. • CN II, III, IV, VI, V_1
Ophthalmoplegia : Restriction of ocular movement d/t CN III, IV, VI palsies	Sequential ophthalmoplegia : CN VI affected → CN III affected → CN IV affected	Concurrent ophthalmoplegia : All nerves affected together → All muscle actions lost at the same time.
Ophthalmic nerve (V_1 palsy) : Corneal anaesthesia	Present	Present

	Cavernous sinus thrombosis	Orbital apex syndrome
Visual loss : D/t CN II palsy.	• No loss initially. • Can be affected in late stages d/t other causes.	• Complete LOV.
Edema in mastoid area : D/t CN V_a palsy	Present	Absent
Chemosis & Proptosis	• B/L • marked/severe (presenting feature clinically)	• U/L • mild
Systemic signs (Eg : fever)	marked	mild
Presentation	Always B/L (starts U/L)	Always U/L.

ORBITAL CELLULITIS

Causes :

1) Extension of paranasal sinusitis (m/c) :
   - Ethmoid sinusitis (m/c).
   - Staphylococcus and Streptococci : m/c etiological agents.
   - m/c in children with upper respiratory tract infection.

2) Exogenous/Trauma

3) Hematogenous spread

Progression :

Pre-septal cellulitis $\xrightarrow{\text{If no } R_x}$ Orbital cellulitis $\xrightarrow{\text{If no } R_x}$ Subperiosteal abscess.

- Findings
  - Eyelid edema.
  - Pain.
  - Inability to open eye.
- R_x : Topical/oral antibiotics.

- Findings
  - Proptosis.
  - Limitation of eye movement.
- Rx : IV antibiotics.
- Complications : Compression of optic nerve ⟶ Blindness.

Note : Orbital septum
- Formed by muscles of eyelid, fascia, medial and lateral canthal ligament.
- Seperates orbit from pre-orbital area forming
  - Pre-septal.
  - Post-septal.

Other Causes & Types of Proptosis 00:37:44

VASCULAR CAUSES OF PROPTOSIS

Carotico Cavernous Fistula (CCF) :

Arterio-venous fistula between carotid artery & cavernous sinus.

Cause : Trauma (75% cases), Spontaneous.

Feature :
- Proptosis :
  - Bilateral
  - Pulsatile
  - Bruit & thrill present, stops on applying pressure to ipsilateral carotid artery.
- Ophthalmoplegia ⟶ First affects CN VI.

IOC : Digital subtraction carotid angiography (DSCA).

Orbital varices :

Congenital enlargement of pre-existing venous channels.

Features : Proptosis

- Unilateral.
- Bag of worms consistency on palpation.
- Non-pulsatile.
- No bruit, no thrill.

Complication : Orbital hemorrhage/thrombosis.

TYPES OF PROPTOSIS

Intermittent proptosis :

- ↑ on valsalva manoeuver or bending forward : Orbital varices.
- ↑ with upper respiratory tract infection : Orbital lymphangiomas.
- ↑ on crying :
  - Children : Capillary hemangioma.
  - Infants : Encephalocele.

Pulsatile proptosis :

- Cortico cavernous fistula.
- Orbital roof fracture in :
  - NF-1
  - Trauma

Pseudo proptosis :

Appearance of eyeball protrusion.

Causes :

- Retraction of upper eyelid : Can be side effect of apraclonidine.
- Enophthalmos : Contralateral eye appears proptosed.
  - Seen in : Trauma, Horner's syndrome.
- Axial length >26mm.
- Buphthalmos (enlarged eyeball).

Thyroid Associated Ophthalmology (TAO)

00:49:38

OVERVIEW

- m/c cause of unilateral and bilateral proptosis in adults.
- Can occur in any state of thyroid activity.

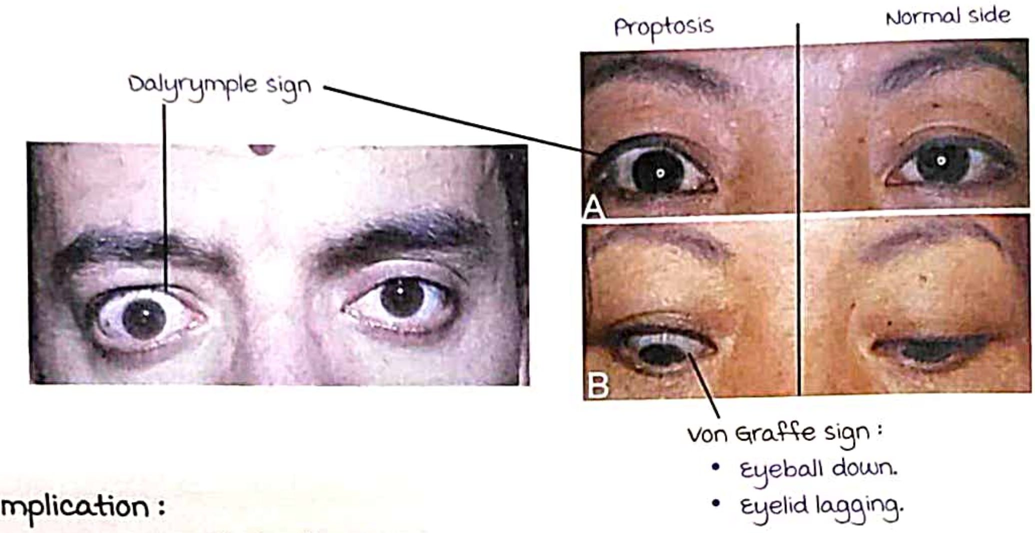
Pathogenesis :

- Inflammation ⟶ Enlargement of extra-ocular muscles.
- Inflammation and inifltration of orbital fat ⟶ Pushing of eyeball forward ⟶ Frank proptosis.

----- Active space -----

CLINICAL FEATURES

	Features
Goldzeiher's sign (earliest)	Congestion/hyperemia.
Dalyrymple sign (m/c)	• upper eyelid retraction ⟶ pseudo proptosis. • D/t constriction of muller's muscle. • Seen in 92% of cases.
Proptosis (2nd m/c)	• Axial unilateral or bilateral. • Seen in 60% cases.
Von Graefe sign	Lid lag : On downgaze, upper eyelid does not move down.
Kocher's sign	Staring/startled appearance.
Stellwags sign	Reduced eyelid blinking d/t upper eyelid retraction.
Restrictive squint	• Fibrosis of extra ocular muscles ⟶ Loss of movement • muscle affected first : Inferior rectus. (Elevation affected first.)



Complication :

Exposure Keratitis (EK) d/t inability to close eye.

Management :

Ensure euthyroid state.

Supportive treatment :

- For EK : Lubricants.
- For upper lid retraction : Botox injection into muller's muscle.
- For inflammation : Systemic steroids.

LACRIMAL APPARATUS : ANATOMY, WATERING EYE AND DRY EYE

Anatomy

00:00:48

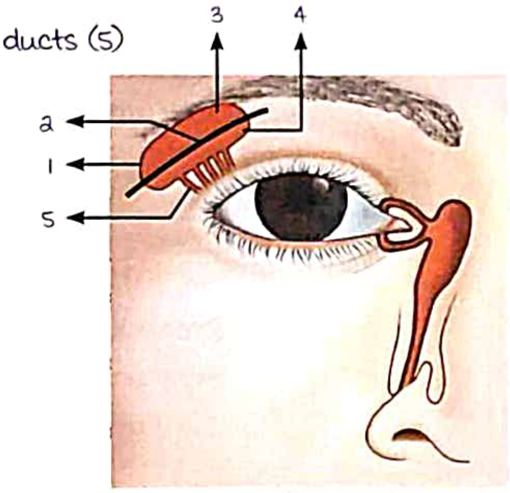
Tears
↓ Secretion
↓ Drainage

SECRETION

Glands :

Main lacrimal gland (1) :

- Divided by levator palpebrae superioris (2) → Orbital part (3) : Superior; → Palpebral part (4) : Inferior
  ↓
  Main excretory ducts (5)
- Function : Reflex tear secretion.



Lacrimal apparatus

Accessory lacrimal glands of Krause & Wolfring :

- Located in fornix (B/w sclera & palpebral conjunctiva).
- Function : Basal tear secretions.

Parasympathetic innervation :

Secretomotor function.

Centre : Superior salivatory nucleus in pons.
↓
Pre-ganglionic N. : Greater superficial petrosal N. (Branch of facial N.)
↓
Ganglion : Pterygopalatine.
↓
Post ganglionic N. : Zygomatic N. (Branch of maxillary branch of trigeminal N.).
↓ Stimulates
Lacrimal gland
↓
Secretion

Sympathetic innervation :

Inhibitory function.

----- Active space -----

DRAINAGE

Puncta (1) : upper & lower.

↓

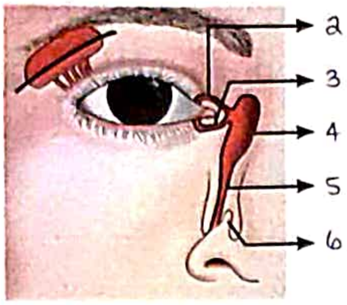
Canaliculi : upper (2) & Lower (3)

↓

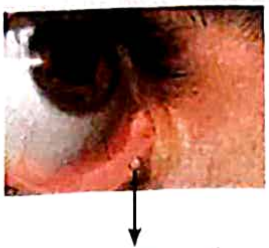
Lacrimal sac (4)

- Surrounded by orbicularis oculi

↓ On blinking



Lacrimal apparatus



Lower puncta (1)

Nasolacrimal duct – NLD (5) opens in inferior meatus of nose (6).

- Opening of NLD guarded by valve of Hasner.
  - m/c block in congenital dacryocystitis.

Watering of Eye & Tumours 00:09:00

Watering of eye → Hyperlacrimation → ↑ tear secretion ← Emotional stress. / Inflammation.

Watering of eye → Epiphora → Blockage of drainage

TESTING OF WATERING EYE

Instruments :

26G bent needle cannula

Nettleship punctum dilator

Bowman's lacrimal probe

Procedure :

Syringing :

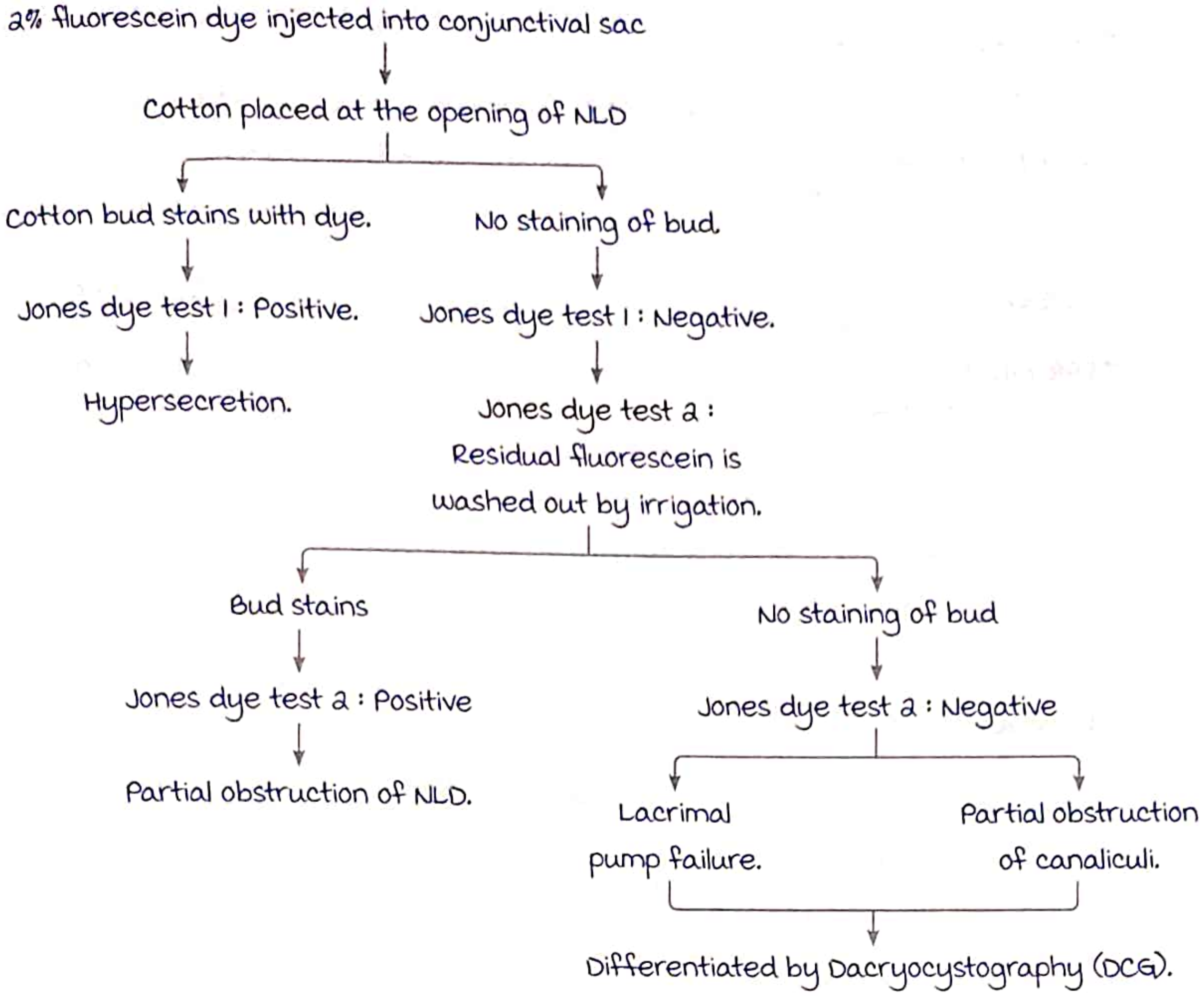
Puncta dilated → Blunt tip of 26G needle inserted into puncta & canaliculi.

↓

Saline injection

- Fluid flows into nose & throat
  - ↓ Patent drainage system
    - Hypersecretion
    - Lacrimal pump failure
    - Partial obstruction
    - (all three) Jones dye test
- Regurgitation of fluid
  - ↓ Obstruction/blockage
  - ↓ Probing
    - Hard stop → NLD blockage
    - Soft stop → Lacrimal sac/ Canaliculi block

Jones dye test 1 :


2% fluorescein dye injected into conjunctival sac
Cotton placed at the opening of NLD
Cotton bud stains with dye.
Jones dye test 1 : Positive.
Hypersecretion.
No staining of bud.
Jones dye test 1 : Negative.
Jones dye test 2 :
Residual fluorescein is
washed out by irrigation.
Bud stains
Jones dye test 2 : Positive
Partial obstruction of NLD.
No staining of bud
Jones dye test 2 : Negative
Lacrimal
pump failure.
Partial obstruction
of canaliculi.
Differentiated by Dacryocystography (DCG).

DACRYOCYSTITIS

Inflammation of lacrimal sac.

Types :

Congenital :

Presentation : c/o watering since birth.

Cause : Non-canalization of NLD.

- m/c site of occlusion :
  Valve of Hasner.

Rx :

- Lacrimal sac massage +
  Topical antibiotics (Tobramycin, erythromycin).
- 9-12 months : Syringing.
- >1 yr : Probing.
- >4 yrs : Dacryocystorhinostomy (DCR).

Acquired :

Cause : Streptococcus hemolyticus.

Rx :

- Oral antibiotics.
- DCR : If fistula (+).

----- Active space -----

TUMOURS OF LACRIMAL GLAND

Pleomorphic adenoma :
Benign tumour

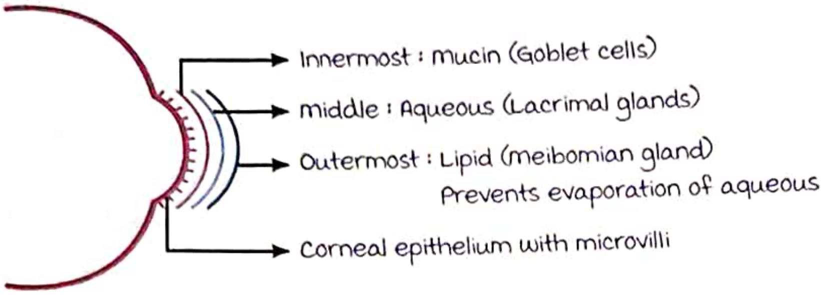
Adenoid cystic carcinoma
malignant tumour

} Displacement of eye downwards and medially d/t location of lacrimal gland.

00:26:45

Dry Eye

TEAR FILM


Innermost : mucin (Goblet cells)
middle : Aqueous (Lacrimal glands)
Outermost : Lipid (meibomian gland)
Prevents evaporation of aqueous
Corneal epithelium with microvilli

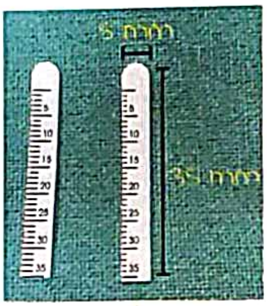
DISORDERS

	Aqueous deficiency	Lipid deficiency	mucin deficiency
AKA	Keratoconjunctivitis sicca	Evaporative dry eye	-
Eg.	Eg. Sjögren syndrome	• meibomian gland disease • Contact lens wearer	Vit A deficiency

TESTS FOR DRY EYES

I. Schirmer test :

- Quantitative test to measure tear production.
- Uses Whatman's filter paper No. 41.


5 mm
35 mm
5
10
15
20
25
30
35

Whatman's filter paper

Procedure :

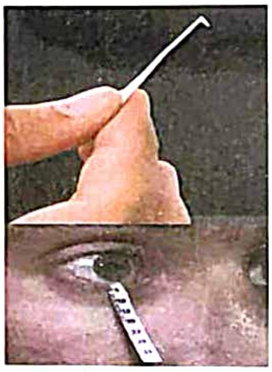
Inserted in lower fornix

↓ 5 mins

Length of wet strip

>15 mm → Normal

<10 mm → Dry eye



Schirmer test

2. TBUT (Tear Film Break-up Time) :

2% Fluorescein instilled in conjunctival sac ⟶ Illuminated with blue light.

↓

Interval b/w a blink & appearance of 1st spot of dryness of cornea

- <10 sec
  ↓
  Mucin deficiency
- Dry spot at a particular location
  ↓
  Corneal disease

3. Phenol red thread test :

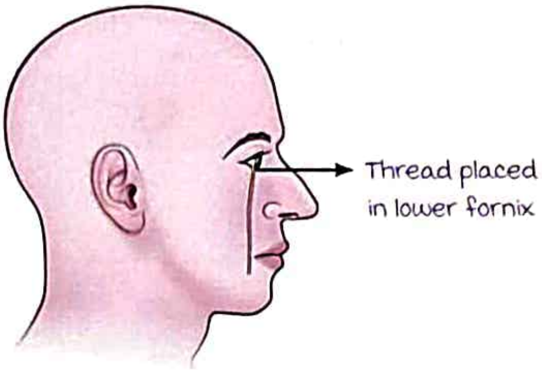
Thread impregnated with phenol red dye :

Yellow in colour

↓ + tears

Red in colour

- <6 mm
  ↓
  Dry eye
- >15 mm
  ↓
  Normal



Phenol red thread test

----- Active space -----

OCULAR TRAUMA

Mechanical Ocular Trauma

00:00:44

Blunt trauma :
- Closed globe injuries.
- Contusions.

Penetrating trauma

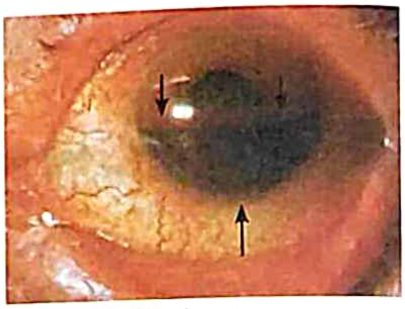
BLUNT TRAUMA

Cause :
- Injury by fist.
- Tennis ball.
- Champagne cork.

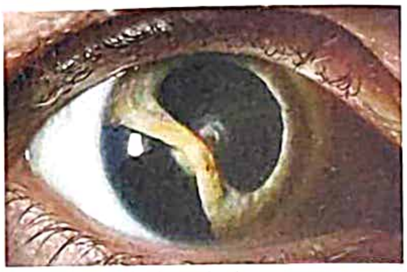
Signs :

1. Black eye : Periocular hematoma.
2. Subconjunctival hemorrhage : Common in trivial trauma.
3. Hyphema : Collection of blood in anterior chamber
   - m/c sign of blunt ocular trauma.
   - Source of bleeding : Circulus Arteriosus major.
   - Rx :
     - Topical steroids : To prevent re-bleeding & associated inflammation.
     - Atropine (Optional).
     - Paracentesis/washing of anterior chamber : If no resorption until 4 days.
4. In Iris :
   - Iridodonesis : Tremulous/trembling iris.
   - Iridodialysis : Detachment of root of iris from ciliary body.

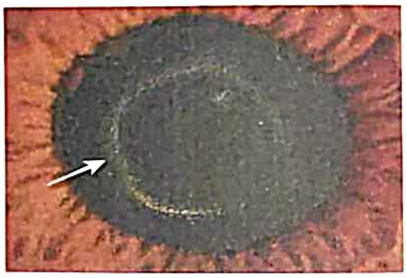
   } D-shaped pupil.
5. In lens :
   - Rosette shaped cataract : Posterior subcapsular.
   - Ectopia lentis/Subluxation of lens :
     - m/c cause : Blunt trauma.
   - Vossius ring : Imprint of miotic pupil on anterior surface of lens.
   - Phacodonesis/tremulous lens.



Hyphema



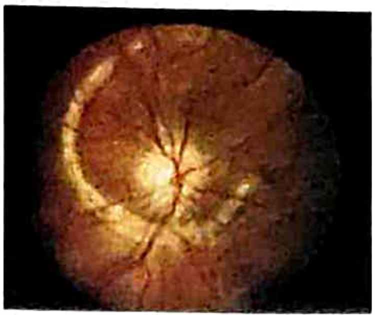
Iridodialysis



Vossius ring

6. Fundus :
- Cherry red spot at macula.
  - Berlin's edema : Fluid collection d/t blunt trauma.
- Commotio retinae : Cloudy grey appearance.
- Traumatic optic neuropathy : Self-limiting.
- Choroidal rupture : Bucket handle configuration.

----- Active space -----



Scleral show d/t choroidal rupture

7. Globe rupture :
- Occurs d/t fall on a knob-like object.
- Site : Supero-nasal limbus near Schlemm's canal.

PENETRATING TRAUMA
- Sympathetic ophthalmitis (Panuveitis).
- Intra-ocular foreign body.

Intra-ocular foreign body :
- Iron, steel : m/c foreign body.
  - IOC : CT scan.
  - C/I : MRI.
- Deposition of iron → Siderosis bulbi.
  - Deposition of rust ring on anterior capsule of lens.
  - Hyperpigmentation of iris.
  - 2° open angle glaucoma.
  - Dx : Prussian blue reaction → Stains iron deposits.

Note :

Ophthalmia nodosa : Contact with Caterpillar hair → Uveitis.

Orbital Trauma

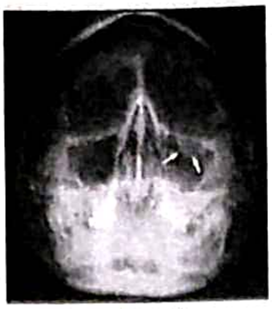
00:24:04

BLOW OUT FRACTURE
- Fracture of floor of orbit.
- m/c fracture in orbital trauma.

Clinical feature :
- Infraorbital nerve anesthesia.
- Double diplopia : Restrictive squint.
  - Fractured bone pieces → muscle entrapment of inferior oblique & inferior rectus → Diplopia in upgaze & downgaze.

Investigation :

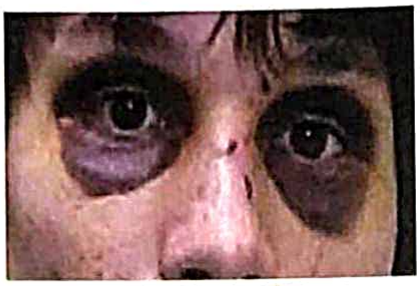
X-ray : Tear drop sign.



Tear drop sign

FRACTURE OF ROOF OF ORBIT

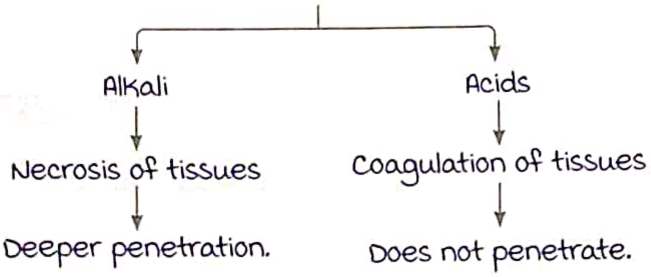
Presentation : Racoon eyes/Panda eyes.



Racoon eyes

Chemical injuries

00:29:49


Alkali
Acids
Necrosis of tissues
Coagulation of tissues
Deeper penetration.
Does not penetrate.

Classification :

Dua's classification.
Roper-Hall classification.
} Grading of ocular surface burns.

Management :

1. Copious irrigation :
   Balanced salt solution/Ringer Lactate by double eversion of eyelids.
2. Topical antibiotics.
3. Atropine : To relieve ciliary spasm.
4. Lubricating eye drops.
5. Topical sodium citrate : Prevents proteolysis.

COMMUNITY OPHTHALMOLOGY

NPCB & VI

00:00:52

Amended from NPCB (Started in 1976) to NPCBVI in 2017.

NATIONAL PROGRAMME FOR CONTROL OF BLINDNESS & VISUAL IMPAIRMENT

NPCB & VI logo

DEFINITIONS OF BLINDNESS

Clinically : No perception of light.
According to NPCBVI (As amended in 2017) and
WHO :

- Presenting visual acuity <3/60 in the better eye with best possible correction.

or

- Limitation of visual field to less than 10° from center of fixation.

Visual Impairment :

Term	Definition according to NPCB & VI
Blindness	Presenting visual acuity <3/60 in better eye with available correction.
Severe visual impairment (SVI)	Presenting visual acuity <6/60 – 3/60 in better eye with available correction.
moderate visual impairment (mVI)	Presenting visual acuity <6/18 – 6/60 in better eye with available correction.
Early visual impairment (EVI)	Presenting visual acuity <6/12 – 6/18 in better eye with available correction.
Functional low vision	• Visual acuity <6/18 or • Visual field <10° from fovea • Able to/potentially able to use vision to plan/execute a task.
Blindness (BCVA/pinhole <3/60)	Best corrected visual acuity <3/60 in better eye.

RECENT FACTS

Prevalence of blindness :

0.36% (2019) according to National Blindness & Visual Impairment Survey (2015-19).

Goal of program :

Reduce prevalence of avoidable blindness (Preventable + curable) to <0.25% by 2025.

Causes and prevalence :

	≥50yrs of age	0-49 yrs
Blindness	• Cataract (m/c) : 66.2% • Corneal opacity due to any cause (2nd m/c) : 8.2%	• Corneal opacity (m/c) : 37.5% • Amblyopia : (2nd m/c) : 25%
Visual impairment	• Cataract (m/c) : 71.2% • Refractive errors (2nd m/c) : 13.4%	• Refractive errors (m/c) : 29.6% • Cataract (2nd m/c) : 25%

Vision 2020

00:17:05

OVERVIEW

- Launched in 1999 at Geneva, Switzerland.
- Adopted by India in 2001 at Goa.

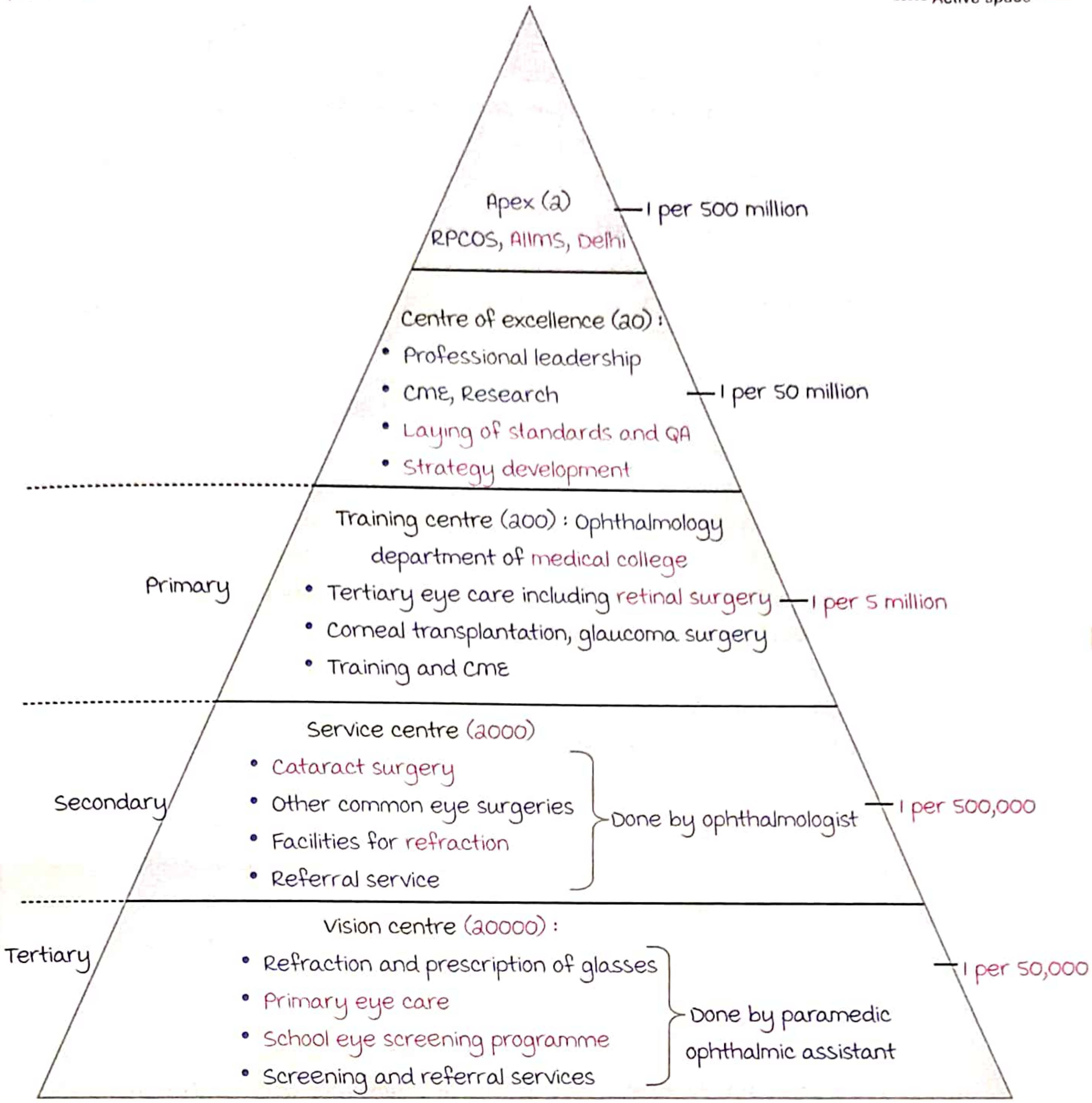
Aims :

- Reduce blind people to 25 million by 2020.
- Eliminate avoidable blindness.

Diseases covered :

India	Globally
• Cataract • Refractive errors • Childhood blindness • Trachoma (Endemic) • Glaucoma • Diabetic retinopathy • Corneal blindness	• Cataract • Refractive errors • Childhood blindness • Trachoma (Endemic) • Onchocerciasis

METRICS PROVIDED BY VISION 2020



Apex (2) — 1 per 500 million
RPCOS, AIIMS, Delhi

Centre of excellence (20) : — 1 per 50 million
- Professional leadership
- CME, Research
- Laying of standards and QA
- Strategy development

Primary

Training centre (200) : Ophthalmology department of medical college — 1 per 5 million
- Tertiary eye care including retinal surgery
- Corneal transplantation, glaucoma surgery
- Training and CME

Secondary

Service centre (2000) — 1 per 500,000
- Cataract surgery
- Other common eye surgeries
- Facilities for refraction
- Referral service

} Done by ophthalmologist

Tertiary

Vision centre (20000) : — 1 per 50,000
- Refraction and prescription of glasses
- Primary eye care
- School eye screening programme
- Screening and referral services

} Done by paramedic ophthalmic assistant

School eye screening programme :

Target : Class V – VIII (10–14 yrs age).

Performed by : Teacher → 1 teacher per 150 students.

Method : Vision screening cards + measurement tape
↓
Record visual acuity (VA)
↓
If VA <6/9 in either eye : Refer to nearest PHC (Primary health centre)

Rashtriya Bal Swasthya Karyakram (RBSK) 00:26:53

Overview

- Launched under National health mission (NHM).
- Screening and early intervention for children (Birth – 18 yrs of age).

SCREENING

Health conditions screened (4 D's) :

4 D's :

- Defects at birth.
- Diseases.
- Deficiency.
- Developmental delays.

Health conditions screened under this program :	
Defects at birth : 1. Neural tube defect. 2. Down's syndrome. 3. Cleft lip & palate/cleft palate alone. 4. Talipes (Club foot). 5. Developmental dysplasia of the hip. 6. Congenital cataract. 7. Congenital deafness. 8. Congenital heart diseases. 9. Retinopathy of Prematurity.	**Deficiencies :** 10. Anaemia especially severe anaemia. 11. Vitamin A deficiency (Bitot spot). 12. Vitamin D Deficiency (Rickets). 13. Severe acute malnutrition. 14. Goiter.
Childhood diseases : 15. Skin conditions (Scabies, fungal infection & eczema). 16. Otitis media. 17. Rheumatic heart disease. 18. Reactive airway disease. 19. Dental caries. 20. Convulsive disorders.	**Developmental delays :** 21. Vision impairment. 22. Hearing impairment. 23. Neuro-motor impairment. 24. motor delay. 25. Cognitive delay. 26. Language delay. 27. Behaviour disorder (Autism). 28. Learning disorder. 29. Attention deficit hyperactivity disorder. 30. Congenital hypothyroidism, Sickle cell anaemia, Beta thalassemia (Optional).
31. Tuberculosis. 32. Leprosy. } Not specific to children.	

mechanism of screening :

Categories	Age group	Estimated coverage	Screening performed by
Babies born at public health facilities (PHC & CHC) and home.	Birth to 6 weeks.	2 crores.	mobile health team.
Preschool children in rural areas and urban slum.	6 weeks to 6 yrs.	8 crores.	Anganwadi & ANM workers.
School children enrolled in class 1st to 12th in government and government aided schools.	6 yrs to 18 yrs.	17 crores.	A school supported by district hospital : Performed via questionaires.

INTERVENTION

1. 0-6 yrs : District Early Intervention Center (DEIC) → Referral to appropriate tertiary centre.
2. 6-18 yrs : Existing public health facility.

LASERS IN OPHTHALMOLOGY

Light Amplification by Stimulated Emission of Radiation (LASER)

Principle :

Laser field → Produce effects in eye

- Photoablation : Burn tissue
- Photodisruption : Break tissue
- Photocoagulation : Coagulate tissue

Types of Lasers

00:02:21

Based on tissue effects caused	Lasers causing photoablation	Lasers causing photocoagulation	Lasers causing photodisruption
mechanism	Producing chemical effects : breaks chemical bonds ↓ vaporizing tissue.	Thermal effect ↓ denaturation of proteins.	3 Effects working simultaneously : • Stripping electrons from nuclei → Ionization of tissue. • Production of plasma-like materials → Breakage of chemical bonds. • Generation of acoustic shock waves → mechanical effect on tissue.
Wavelength used	Ultraviolet (150nm – 300nm)	Visible spectrum (400nm – 580nm)	Infrared : invisible ↓ Helium-neon beam (red) used to view and aim beam.
Examples	Excimer laser: Argon & Fluoride (193 nm)	• Argon green laser: 514nm • Frequency doubled Nd: YAG laser : 532nm • Diode laser : 810 nm (preferred in retinopathy of prematurity). • Ruby Red laser : 694nm	• Nd : YAG (Neodynium Yettrium : Aluminium Garnet) : 1064nm. • ND : Glass : 1053nm.
Uses	Corneal refractive procedures : • LASIK • PRK	• Pan retinal photocoagulation for: – Diabetic retinopathy – CRVO • Laser trabeculoplasty for POAG. • Focal retinal laser photocoagulation for: – Diabetic macular Edema – CSR – ARMD	Nd: Glass : • FLACS for Cataract sx. • SMILE for refractive error correction. Nd: YAG: • Posterior Capsulotomy for PCO. • Peripheral iridotomy for ACG.

PHYSIOLOGY OF VISION

Visual pigment :

Rhodopsin ↓ Components

Opsin/Scotopsin :

- G-protein coupled receptor (GPCR) ↓ Enables signalling.

11-cis retinal :

- Light sensitive chromophore $\xrightarrow[\text{Transduction}]{\text{Absorbs light}}$ 11-trans retinal (more stable isomer)

Visual Cycle

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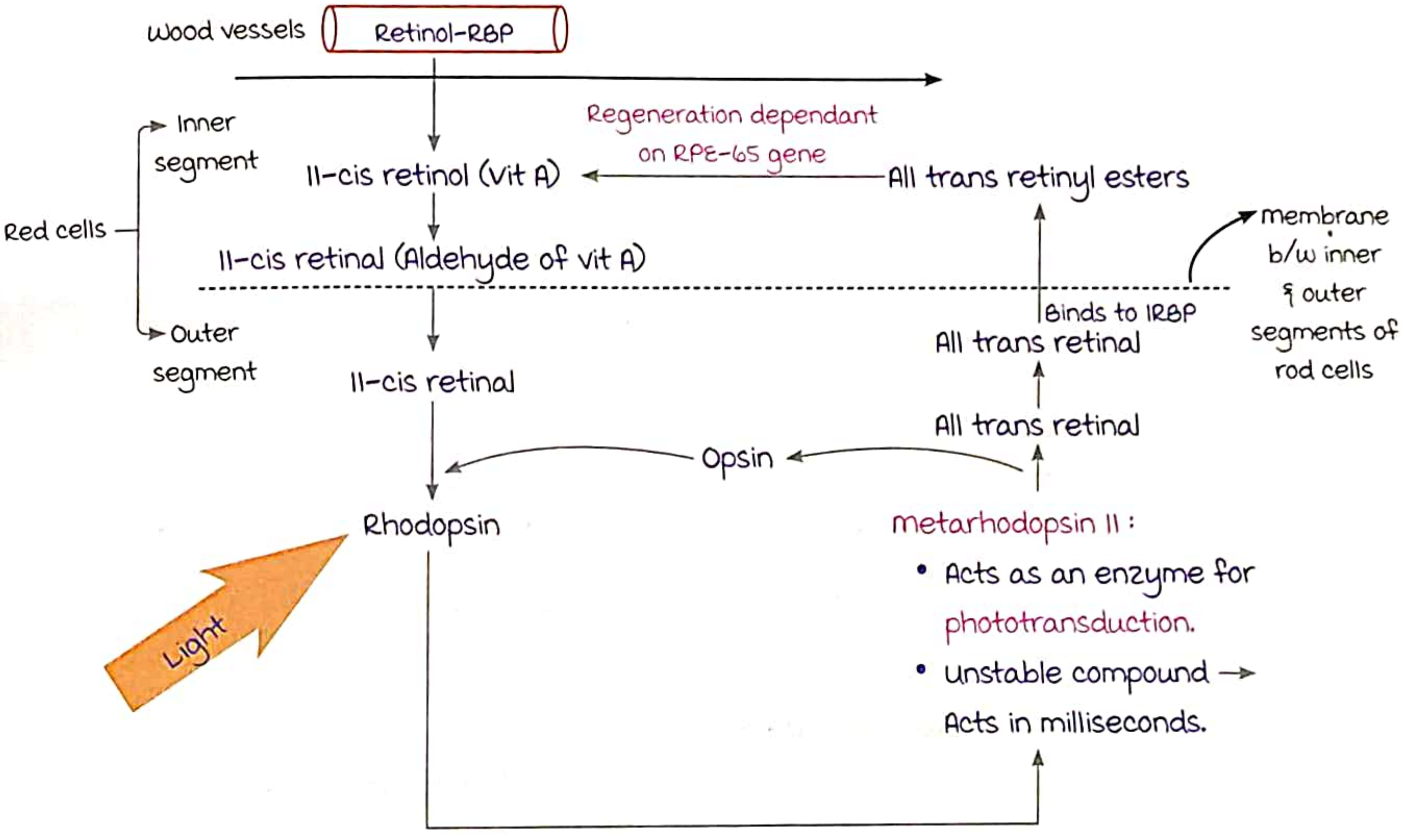
Site : Photoreceptors (Rod cells).

RBP : Retinol binding protein ↓ Binds retinol in blood.

IRBP : Intra Retinal Binding Protein.

PATHWAY


Blood vessels
Retinol-RBP
Inner segment
Red cells
Outer segment
11-cis retinol (vit A)
Regeneration dependant on RPE-65 gene
All trans retinyl esters
11-cis retinal (Aldehyde of vit A)
membrane b/w inner & outer segments of rod cells
Binds to IRBP
All trans retinal
11-cis retinal
All trans retinal
Opsin
Rhodopsin
Light
metarhodopsin II :
• Acts as an enzyme for phototransduction.
• unstable compound → Acts in milliseconds.

----- Active space -----

PHOTOTRANSDUCTION & HYPERPOLARISATION

metarhodopsin II

↓

GDP → GTP exchange

↓

Activates transducin (Responsible for transduction)

↓

Activates PDE enzyme

↓ catalyses

cGMP $\xrightarrow{\text{Hydrolysis}}$ 5'GMP

↓ cGMP concentration :

- Na⁺ channels close (cGMP responsible for opening Na⁺ channels).
- Na⁺ ion influx stopped.
- Glutamate efflux stopped.

↓

Hyperpolarisation :

- Graded to local potential (Not an action potential).
- Transmitted by electronic conduction.

↓

Reaches bipolar cells

↓

Reaches ganglion cells : Site of action potential.

↓ Via visual pathway

Lateral geniculate body (LGB)

↓

6 layers

Layer 1 and 2 :
magnocellular pathway
↓
Responsible for :
- motion.
- Black & white vision.

Layer 3 to 6 :
Parvocellular pathway
↓
Responsible for :
- Fine details.
- Color vision (Photo transduction).

- Under light → Hyperpolarising potential created.
- Under dark → Depolarizing state.

DEFECT IN VISUAL PATHWAY

RPE-65 gene mutation → No regeneration of 11-cis retinol (Chromophore) —causes→
- Retinitis pigmentosa.
- Leber's congenital amaurosis.